HISTOPATHOLOGICAL INVESTIGATIONS OF THE DENTAL GRANULOMA[1]

A Preliminary Report

NATHANIEL FREEMAN, D.D.S.

Adjunct Dentist, Mount Sinai and Montefiore Hospitals, New York City

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I. DEFINITION

The examination of the tissues around the root-ends of teeth having dead pulps is of interest to all who are concerned with the problem of focal infection. The microscope gives us a clear picture of the reaction of the tissues to the infection that emanates from the root canal. We must have a thorough understanding of the definition of a dental granuloma before we discuss its histopathological phases. The term granuloma, as applied here, is a misnomer. Some authors are disposed to class together, under the name "*infectious granuloma*," lesions induced by microörganisms, certain phases of which are characterized by a localized, usually slow, nodular growth tending to necrosis. *Infectious granulomata* are pathologically situated between pure inflammation and neoplasms. They are differentiated from neoplasms such as sarcoma, etc., by the fact that they contain many dissimilar types as fibroblasts, polymorphonuclear leucocytes, plasma cells, etc.

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A dental granuloma is a formation of new inflammatory tissue. It is a chronic inflammatory lesion caused by a mild injurious agent that produces proliferation of the fixed-tissue cells of the peridental membrane, and infiltration of lymphocytes and leucocytes. The lesion grows at the expense of the bone, which therefore is always more or less involved. It is probable that, if a similar lesion could be reproduced in a non-osseous environment, it would be flat; and if it could grow in an area where it would meet no resistance, and if its original point of development were not a minute opening, such as a root foramen, it would present nothing more than a connective-tissue proliferation.

The term "*pericementoma*" has been advocated by Pollia as being more descriptive. The only objection to this name is the desire on the part of the students of neoplasms to confine the termination "oma" to new growths of non-infectious origin.

II. DIFFERENTIATION

A granuloma is often described as a "*blind abscess*," but this is incorrect. "Abscess" implies a circumscribed collection of pus in a cavity formed by the disintegration of tissues and usually surrounded by a wall of lymph. An *acute alveolar abscess* is a suppurative inflammation, and involves a process of destruction of the peridental tissues. Unless the cause is removed, it resolves itself into a *chronic* alveolar abscess. An acute abscess usually starts with symptoms of violent inflammation, whereas the formation of a granuloma begins and continues without the patient's knowledge.

III. ETIOLOGY

[As the histologic changes are those of a sub-acute inflammation, the cause must be looked for in an organism of low virulence, such as *Streptococcus viridans*. The lesion is formed by a reaction of the tissue to mild injurious agents, such as bacteria in small numbers; toxins, caused by bacterial activity in pulp chambers; and chemicals, such as formaldehyde used in root-canal treatment.] Many interesting theories have been advanced as to the formation of the granuloma.

Greve's theory. In gingival tissues there are many embryonal rests of epithelial cells, left in the course of the development of the tooth

germ. If, in the neighborhood of such a rest, the pulp of a given tooth breaks down, granulations form on the outside of the tooth, which enclose either certain lipoid-containing cells (so-called "foam" cells) or some of the embryonal epithelial rests. The centers of these areas of proliferation then undergo degeneration and liquefaction, and a cyst or a granuloma forms.

Röhrer's theory. Granulomata are of embryonic derivation, but there is a constitutional predisposition to cyst formation. This predisposition becomes apparent when the area is subjected to insult, as by caries, luetic infection, and blows.

In our series of cases we have found two types: (*a*) Those surrounded by a *fibrous capsule*, without the presence of epithelium; (*b*) and those containing *epithelium* in the form of network throughout the tissues.

IV. DIAGNOSIS

Diagnosis is usually made by roentgen examination. In fact it was the first noted of the radiographic variations, since its appearance is the most striking of all dental abnormalities, except the dentigerous cyst and the dental tumor. It appears as a dark, localized, circumscribed area about the apex of a tooth. It is seen as a dark area with a distinct white line about it. If the process of destruction involves the cancellous bone only, then the area will be hazy in appearance. If one or both plates of bone are involved, then the radiograph appears very dark owing to the greatly lessened density.

V. SIMPLE DENTAL GRANULOMA

The reaction caused by the injurious agent consists of a proliferation of fibroblasts and vascular endothelium of the peridental membrane. The part nearest the source of injury becomes infiltrated with lymph and a small number of leucocytes. This focus becomes surrounded by a fibrous layer, which is continuous with the peridental membrane. There is an increase in the number of cells and the formation of plasma cells. The lesion gains in circumference, and usually attains the size of a pea. At this stage the simple granuloma is surrounded by the fibrous capsule, which extends into the medullary spaces between the trabeculae of bone. This is the reason why the

granuloma is sometimes retained in the jaw when the tooth is extracted (*fig. 1*). The thickness of the capsule varies greatly. Fibers are arranged in bundles and grow in various directions. The vessels and capillaries are abundant among the fibers of the capsule. Their lumen is often greatly increased and contains red cells, a granular substance, and occasional polymorphonuclear leucocytes. The endothelium often is proliferative; and contains fibroblasts and plasma cells. The

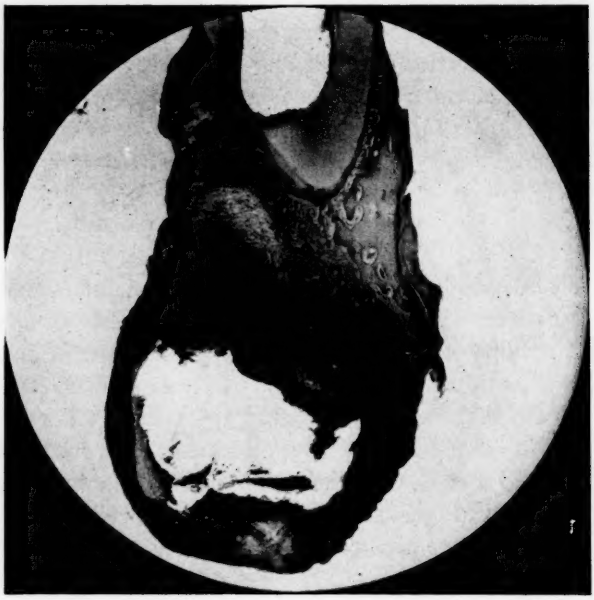

FIG. 1. Upper central showing large simple granuloma. Radiographic diagnosis was that of cyst.

capsule seems to represent an effort by nature to wall-off the seat of inflammation, to prevent its spreading into neighboring parts. This capsule, however, does not prevent absorption, as it contains a network of capillaries and is penetrated abundantly by larger vessels, thus establishing direct communication between the inner part, the seat of inflammation, and the circulation. Bacteria and toxins may therefore be carried away by the blood stream. The inside of the granuloma appears at first to be a great mass of plasma cells (*fig. 2*),

but one also sees capillaries, which are often lined by only a single layer of endothelium, and occasionally are extremely dilated and crowded with blood cells. These plasma cells, obviously derived from the lymphocytes through a modification of the nucleus and protoplasm,

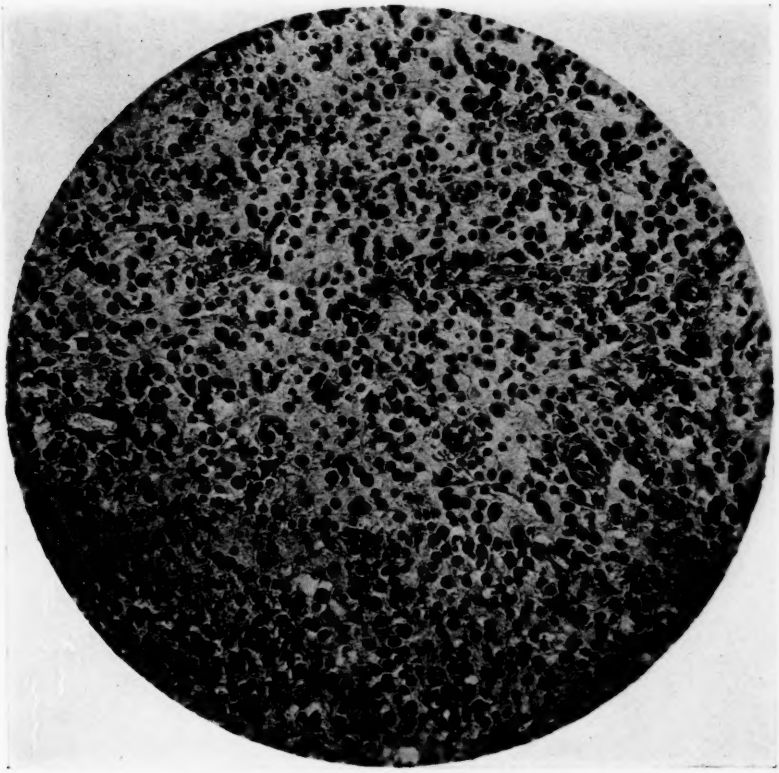

FIG. 2. Area in granuloma showing marked plasma-cell infiltration.

are rather larger than lymphocytes, and usually of a rounded or oval form with the nucleus eccentrically placed, generally at one end of the cell.

One or two places in granulomas may show necrosis—a certain number of cells are detached from the rest, and more or less broken down. Polymorphonuclear cells are then found in great numbers in

the capillaries, and in the tissue where the process of destruction is going on (*figs. 3* and *4*). The pus that is formed may be absorbed, halting the infiltration of leucocytes and leaving a space or spaces in the central part of the granuloma. Such a space is called a lumen,

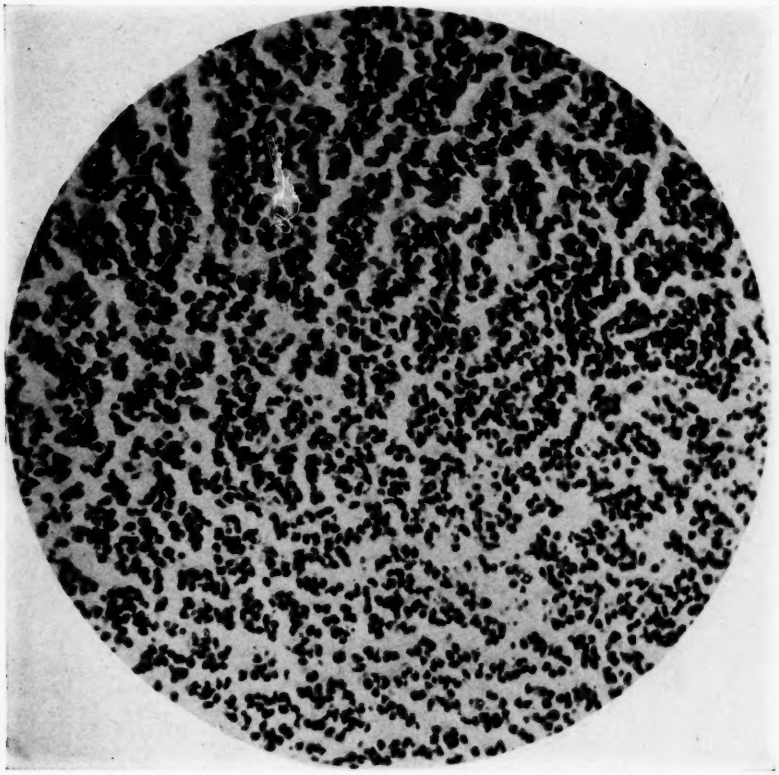

FIG. 3. Area in granuloma composed almost wholly of polymorphonuclear leucocytes (pus cells).

and may contain remnants of the decomposed tissue as well as inflammatory excretions from the pulp canal or remaining part of the lesion. The process of suppuration is usually caused by bacteria that pass from the root canal, or are forced into the granuloma by instrumentation. The pus is usually taken care of by the defensive forces of the

body, which cause its absorption. Occasionally, however, the destructive process becomes more extensive, tearing down the fibrous encapsulation and forming a sinus to the surface of the gum.

There is also evidence of cholesterol formation, especially in old specimens (*figs. 5* and *6*). These cholesterol crystals are frequently

FIG. 4. Section of granuloma showing central portion undergoing necrosis.

surrounded by foreign-body giant-cells, but in this work these cases were rare. Occasionally fatty degeneration occurs, usually at the periphery just inside the capsule. Hyaline, found quite frequently, accumulates in droplets of various sizes in the cytoplasm of the plasma cells. Often the entire cell is so loaded with hyaline that

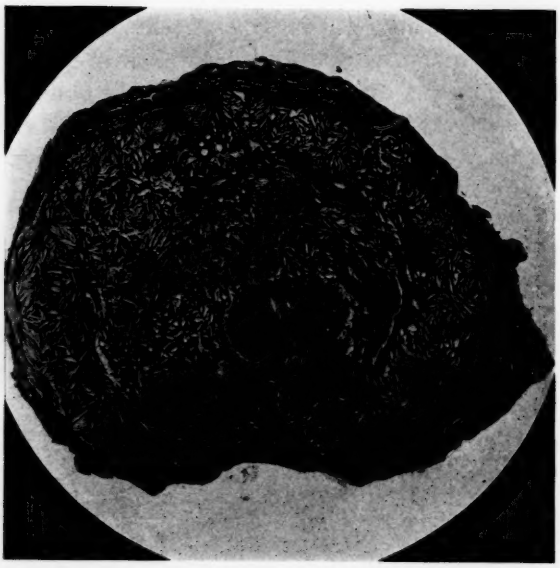

FIG. 5. Epitheliated granuloma, probably of long duration, showing cholesterol formation. See *fig. 6*.

FIG. 6. Higher magnification of portion of *fig. 5*.

the nucleus is pressed to one side, and nothing remains of the cell but the acidophilic hyaline. These cells are called "*Russell bodies*" (*fig. 7*).

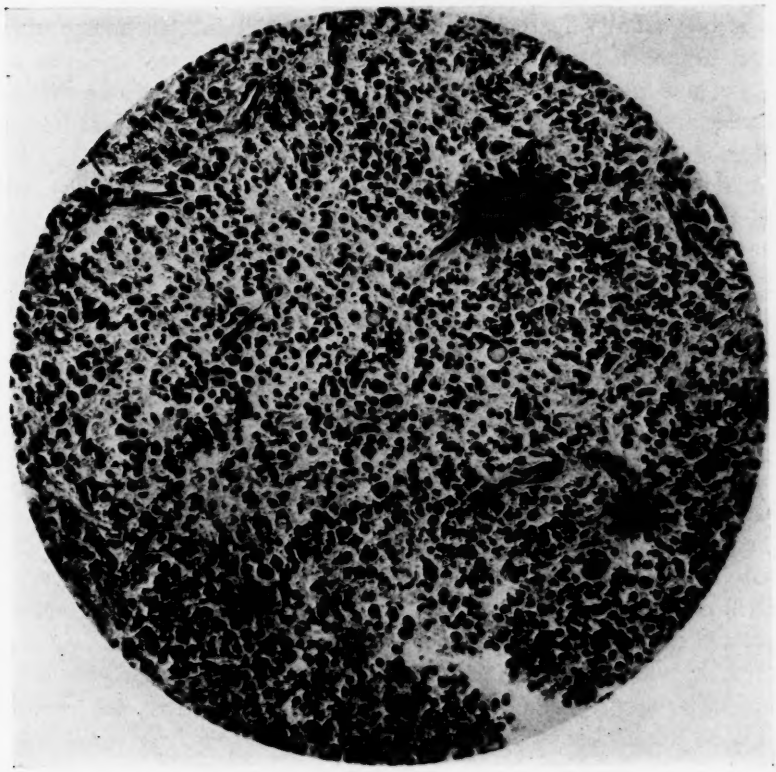

FIG. 7. Russell fuchsin bodies in granuloma; scattered throughout entire field. Note also large number of plasma cells.

VI. RUSSELL FUCHSIN BODIES

The occurrence of numerous Russell fuchsin bodies in these granulomas was most interesting. We found them present in 80 to 85 per cent of our cases. Because of the confusion in the literature regarding the so-called Russell fuchsin bodies, it may be well to give the generally accepted description. These bodies are round or oval on section, varying in size from that of an eosinophile granule to two or three times

the diameter of a red blood-cell. They are highly refractile and sharply contoured, occurring singly or more commonly in clusters of variable sizes both intra-cellularly and extra-cellularly. They showed a strong affinity for acid stains. The Russell fuchsin bodies were first described by Fox in 1858. In 1890 Russell described them as organisms characteristic of cancer. While studying the growth of cancer in various organs, Russell first noticed round or oval bodies that stained with fuchsin and iodine green. Dubreuil and Favre brought out the fact that although the Russell fuchsin bodies are regarded as products of degeneration, yet their presence is not always accompanied by signs of degeneration. Nothing is known concerning the genesis of these bodies except that they appear in chronic inflammatory processes. Among the many theories advanced to explain their origin and nature, two have become generally accepted: (*a*) their origin from cell granules, especially those of plasma cells, mast cells and leucocytes; (*b*) their origin from red blood-corpuscles.

Brown said that there is no single pathologic condition or class of conditions in which fuchsin bodies occur exclusively, although they are frequently found in tumors, rhinoscleroma, granulation tissue, plasma cells, and connective-tissue cells. While their occurrence within cells other than plasma cells is comparatively rare, typical fuchsin bodies can be found in other types of cells. It has been pointed out that the areas in which they occur most numerously are those in which granulation tissue, plasma cells, and newly formed capillaries occur. Von Mueller emphasized the fact that the genesis of these Russell bodies varies, and stated that they may arise not only from plasma cells but from other cells.

There is reason to believe that these bodies increase in number by direct division as soon as they become extra-cellular. This peculiarity has led many observers to regard them as fungi. Under high magnification, the Russell body appears to have a clear space surrounding it. If the Russell body is the result of a degenerative process, then one would expect to find other evidence of degeneration where these are formed, but this is frequently far from the truth. While there is not always a relationship between fuchsin bodies and degenerations, their distribution in various tissues shows a definite relation to blood

vessels, to red blood-corpuscles and to pigment derived from them, and to pigment of non-hematogenous origin.

In some areas the connective-tissue cells and fibres undergo degeneration, the cells becoming foamy (so-called "foam" cells) and re-

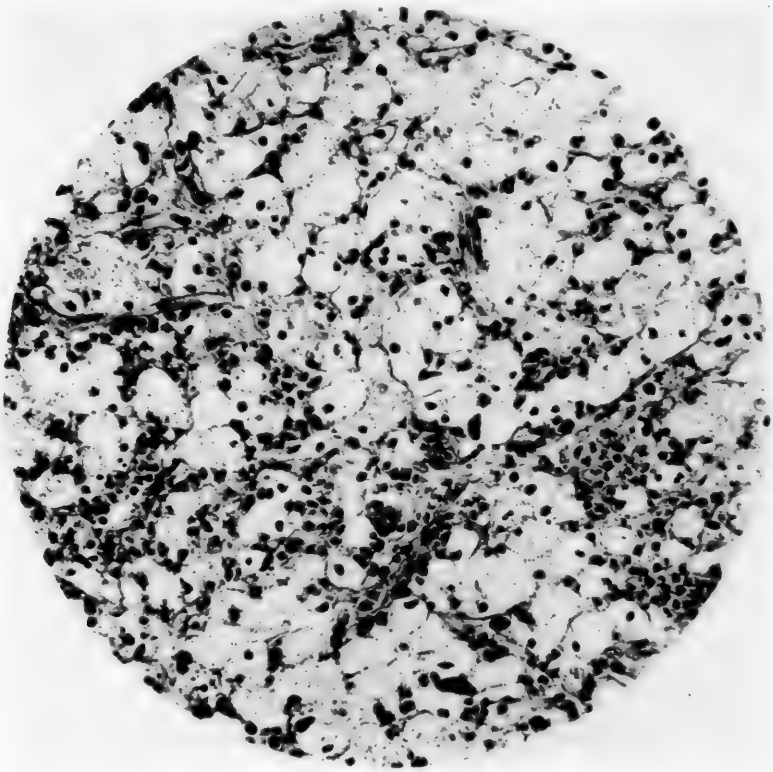

FIG. 8. Section of granuloma showing area of so-called foam cells.

sembling those of the xanthoma of other regions of the body. These cells represent a connective-tissue reaction following an assembly of cholesterol esters in the cells of the affected site. The cells usually contain one or more darkly staining nuclei. The granules in these cells are composed of combinations of cholesterol and fatty acids as esters (*fig. 8*).

Foreign-body giant-cells have been observed, in this work, but these have been rare in contradistinction to the findings of other observers.

VII. EPITHELIATED DENTAL GRANULOMA

Another interesting feature in these granulomata is the presence of a squamous epithelium. Their cells originate in the embryonal rests, or remnants of the primary enamel organ. In the great majority of cases, these epithelial structures are normally present in the peridental membrane. To understand the origin of these groups of cells,

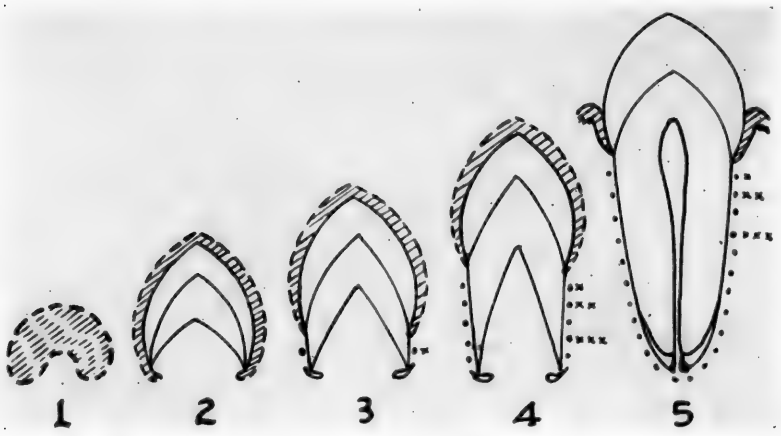

FIG. 9. Diagram illustrating development of epithelial rests during growth of root. *X* shows position of epithelial rests. (After Orban)

it is necessary to refer to the development of the enamel and enamel organ (*fig. 9*). The enamel organ is developed by epithelium, the remainder of which covers the enamel during the whole developmental process up to the time the full anatomical crown is erupted. In a tooth germ in which the formation of dentin and enamel has begun, we see at the apical end of the tooth germ the outer enamel epithelium passing over into the inner layer and there forming a loop. This loop is called Hertwig's sheath (*fig. 10*). The epithelium of the enamel does not end where the enamel ends, but enamel epithelium is also found during development of the root dentin at its apical region, up

to the time the apex is fully developed. A continuous epithelial sheath exists only at the beginning of root formation. As development progresses, and the root elongates, the connection between Hertwig's epithelial sheath and the enamel is severed. The enamel organ ends with the enamel, and Hertwig's epithelial-sheath remains as a

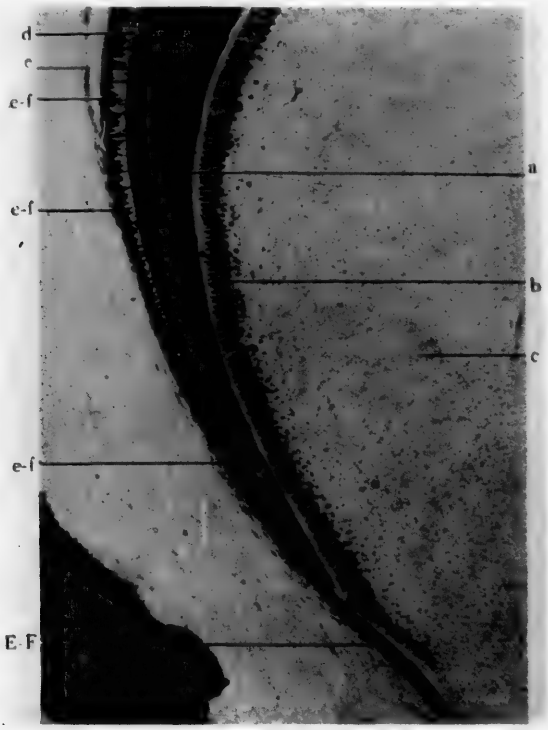

FIG. 10. Hertwig's epithelial sheath (*E–F*) is continuation of outer (*e*) and inner (*f*) enamel epithelium. (*a*) dentin, (*c*) pulp, (*d*) enamel. (After Orban)

loop on the end of the root separated from the enamel epithelium. The epithelial rests remain between the enamel organ and Hertwig's epithelial-sheath, indicating the former continuous connection. As soon as the dentin is fully developed, the function of the epithelial sheath is completed, and it disappears, leaving only epithelial rests

to indicate its former existence. In growing teeth, the epithelial rests are found in the inner layer of the peridental membrane.

So long as there is no irritation, the epithelial rests remain quiescent indefinitely. If, however, an infection occurs, there is then a reaction on the part of all the cells. As the chief property of squamous epi-

FIG. 11. Island of epithelial tissue within granuloma.

thelium is to act as a protective covering, the isolated cell nests undergo proliferation and there arise dividing and branching chains of epithelium, which grow like a network through the lesion. The cells of the proliferating epithelium differ in appearance from the cells of the epithelial rests; they become large, and the cytoplasm and nu-

cleus become more distinct (*fig. 11*). When necrosis occurs in epitheliated granulomas, the epithelial strands (*fig. 12*) have a tendency to grow between live and necrosed tissues, sometimes giving the appearance of an endeavor to encapsulate the tissue that has been destroyed. Such epithelial linings, which may actually cover the

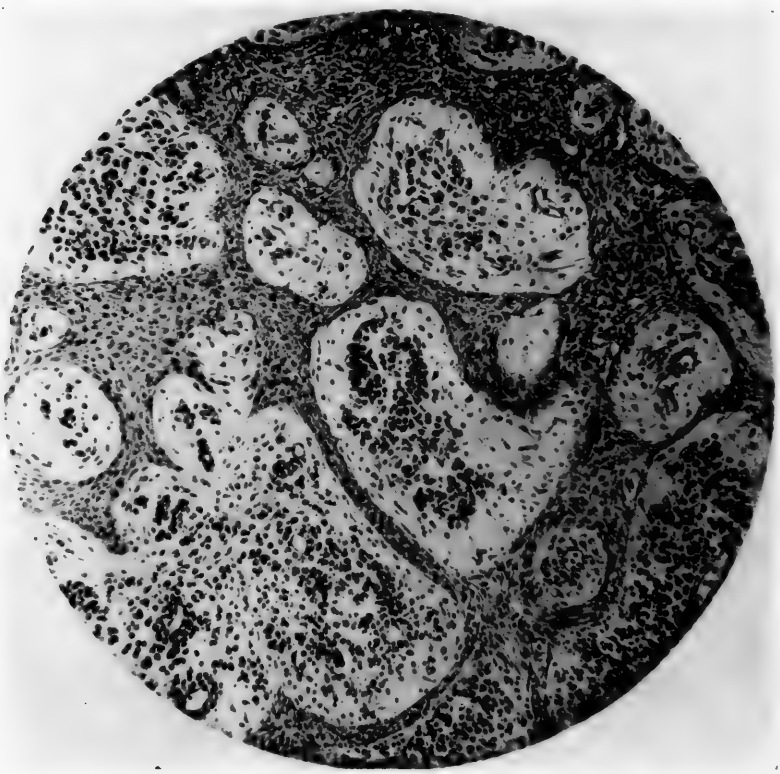

FIG. 12. Section of granuloma showing network of proliferated epithelium.

entire inner surface of the lumen of a granuloma, must be regarded as the beginning of a periodontal or radicular cyst (*figs. 13, 14,* and *15*). The exudates accumulating in the lumen cause extension of the cyst, which may grow to the large size with which we are clinically familiar. Careful study of the history and pathology of this type of cyst shows

FIG. 13. Section of upper molar showing small epitheliated granuloma.

FIG. 14. Section of upper molar with small cyst attached to buccal roots. Radiographic diagnosis was granuloma.

that it is infectious in origin. X-rays also show that such cysts are almost always connected with devitalized teeth. As the lining of these cysts is a squamous epithelium, they sometimes have been regarded as dermoid in nature. Occasionally the lining is partly or wholly of columnar cells, which indicates their paradental origin.

FIG. 15. Cyst wall in granuloma showing necrotic tissue.

Recent investigators have claimed that all granulomata contain epithelium. Our series of cases have disagreed with this claim. Approximately 45 per cent of the granulomata were devoid of any epithelial tissue. It is difficult to explain why in one case epithelial tissues predominate in the apical areas and in another case connective tissues.

It is very likely that living conditions in one case are more favorable for epithelium; in another, for connective tissue.

We have not gone into the bacteriological phases of granulomata because it has been demonstrated that bacteria are present, being demonstrable both in section and in culture.

VIII. SUMMARY

As a preliminary survey, over two hundred dental granulomata at the apices of teeth were examined histopathologically. Of these, at least 45 per cent were found to be devoid of any epithelial tissue. There is definite histological evidence that the epithelium present in some of these granulomata is derived from the remnants of the epithelial sheath of Hertwig.

The presence of Russell fuchsin bodies has undoubtedly some definite significance, possibly as degenerative types of plasma cells and diagnostic of chronic productive inflammation of this specialized tissue.

From the standpoint of focal infection there is undoubtedly no question that bacteria or their toxins are not limited by the fibrous capsule. Histopathological evidence, in the form of a capillary network in the capsule, shows that there is a direct communication between the inner part and the circulation.

Since epithelial rests have been demonstrated in normal peridental tissue, and it is impossible to differentiate clinically or radiographically between a simple granuloma and epitheliated forms, the removal of all granulomata is indicated from the standpoint of therapy.

The author wishes to express to Dr. A. Furedi, of Dr. Theodor Blum's Laboratory, his sincere appreciation of the preparation of some of the pathological slides.

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DISCUSSION

Joseph Schroff, M.D., D.D.S. (*New York City*): The topic, "histopathology of the dental granuloma," chosen for this discussion, indicates that the subject of granulomata is not a closed book. As the essayist mentioned in his introduction, granulomata and focal-infection problems are closely related. Those who heard Dr. Pollia last month[2] realize that the problem of focal infection is far from being solved. The value of any investigation in this field is directly proportional to the amount of actual knowledge acquired, whether positive or negative, that can be applied therapeutically. Of course one might say that, if we were to prevent dental granulomata from forming, the problems of the granuloma and focal infection would be eliminated. But until that day comes, there will be need for continued investigation. The purpose of Dr. Freeman's paper is two-fold: (*a*) to review the present knowledge of the dental granuloma, and to disseminate particularly some of the lesser known facts about its histopathology; and (*b*) to present any additional or confirmatory facts resulting from his investigation.

Except for the small percentage of cases of chronic diffuse apical periodontitis, the dental granuloma is the commonest form of chronic apical periodontitis that the average man observes in practice. By many the

[2] Pollia: *Journal of Dental Research*, 1931, xi, p. 83.

term "granuloma" is considered a misnomer. To me, the term does not seem unsuitable. It was a fortunate choice, because the word "granuloma" represents a type of tissue reaction that occurs in other parts of the body. This fact serves to show that dental pathology is nothing but general pathology as it is modified by the particular structure or tissue involved—in this case, the tooth with its peridental membrane. In medicine the term granuloma is applied to chronic inflammation of tissue in tuberculosis, syphilis, or leprosy, and to that in gum tissue. To the pathologist the term conjures up microscopic pictures having certain features common to all, namely, a more or less loose reticulated fibrous structure, containing endothelial and reticular cells; and a cellular infiltration consisting of small lymphocytes, and plasma cells, with varying numbers of multinucleated large cells; and some leucocytes, depending upon any attendant secondary infection. As Dr. Freeman indicated, a dental granuloma is caused by a low-grade infection or trauma, which slowly destroys the pulp and activates the periapical portion of the peridental membrane to a defensive, proliferative tissue-reaction, in the form of a circumscribed local mass of granulation tissue. The microscopic picture corresponds in most respects with that seen in the conditions mentioned above.

The better known constituents of the granuloma. The young granuloma, if examined microscopically, has the characteristic architecture seen in granulomata of gum tissue, consisting of a loose reticular syncytial stroma in which there are reticular cells, endothelial cells, and large numbers of lymphocytes and plasma cells. These lymphocytes and plasma cells first assemble around the blood capillaries but later appear in the fixed tissues. Leucocytes may also be found, the number depending upon the infection present. At the periphery the fibrous tissue is arranged in a circumferential manner, which gives the appearance of a capsule. As the granuloma is subject to constant infection and re-infection from the infected dental tubuli of the tooth, the typical picture changes and becomes more complex. Usually there is a marked increase in the number of leucocytes.

The lesser known constituents of the granuloma. The Russel bodies and "foam" cells are probably not so well known to the dentist. These cells have been recognized for a long time. Their significance has invoked a great deal of heated discussion. At present, in the prevailing opinion, they are not important. They are not forms of degeneration, but rather represent the index of protoplasmic ability of the cells to take up and store within themselves the by-products of degeneration—the result of the battle between bacteria and the defensive cells in the granuloma. The Russel bodies are considered to be derived from the plasma cells. The "foam" cells represent

fatty infiltration and not fatty degeneration. This fatty infiltration is present in the giant cells or histocytes or macrophytes (they are practically the same thing). The fatty substances in the granuloma are produced by the destructive action of the leucocytes against bacteria. The leucocytes liberate proteolytic substances. The by-products of destruction are ingested by the foreign-body giant cells, and are stored up in the cells, this infiltration of fatty by-products suggesting the presence of foam in cross sections of the cells. Excessive amounts of fatty substances in the cell bodies tend to rupture the cell membrane, spilling the fat into the surrounding tissue. There the free fatty-substances tend to crystallize and form the cholesterol crystals that occur in some granulomata. Other giant cells in turn try to get rid of these crystals.

The practical importance of knowing whether all granulomata are epitheliated. Until recently granulomata were classified as either epitheliated or non-epitheliated, the findings of most investigators having shown that approximately 50 percent are epitheliated. Dr. Freeman's results accord with them. However, the recent findings of Dr. Hill of Western Reserve Dental School are distinctly different and of great importance.[3] In Dr. Hill's study, based on a serial-section microscopic examination of forty-two dental granulomata, all were epitheliated. Further investigations are necessary. The only studies of value are obviously those of serial sections. We all know cases of teeth with areas of decalcification at the apex considered to have been granulomata, which, after proper root-canal therapy, were supposedly cured. (X-rays taken at later dates in these cases apparently showed the areas of decalcification to have been recalcified.) I believe that such cases consist of non-epitheliated granulomata. It is well known that epithelial cells prevent or interfere with healing, or are an ever-present source of danger in tissue as cell remnants. Of course, microscopic sections of infected cyst membranes often show that the epithelial lining of such cysts becomes necrotic and sloughs away, so that at times it is difficult to find even traces of epithelial cells. If all granulomata contain epithelial cells, then the only explanation for their cure is that the drugs used in the root-canal therapy have brought about the destruction of the contained epithelial cells.

Do granulomata heal themselves? I believe that some granulomata heal themselves. Unfortunately, the number of observations, on a large series of patients with untreated dental granulomata, has not been sufficient to decide. Most of our observations have been on patients who have had

[3] Hill: *Journal of Dental Research*, 1930, x, p. 323.

systemic expressions of focal infection, or have suffered from some other disease, and whom it was considered inadvisable to use for experimental purposes. Hence, such patients were invariably advised to have such teeth either extracted or root-canal treatment instituted.

Microscopic studies of the structure of a large number of granulomata show wide differences in detail. The microscopic picture of an old granuloma differs from that of a young one. In older granulomata, especially those attached to teeth with apical foramina closed against re-infections from the dentinal tubuli, the cellular elements contain no leucocytes, fewer plasma cells, and a preponderance of lymphocytes. Instead of a loose type of stroma, we see more fibrosis and scar tissue; and in addition, cholesterol spaces and multi-nucleated giant cells—evidence of arrest of bacterial activity and healing. In some granulomata calcification and ossification have been found.

Granulomata theoretically are a defensive mechanism produced in the tissue to wall-off bacterial invasion into the blood stream. At times the defensive powers of the granulomata are inadequate. Regardless of the fact that some granulomata heal themselves, as shown by microscopic evidence, it is advisable, under present conditions of insufficient knowledge, to recommend that patients with dental granulomata should have some therapy, either root-canal to be followed by root resection, or extraction of the tooth with the granuloma, as indicated.

I desire to commend Dr. Freeman for the work he has done, and wish to add my word of encouragement for further studies. I realize the handicaps under which he has been laboring.

Jacob B. Schneer, D.D.S. (*New York City*): While the histopathological pictures as shown by Dr. Freeman represent a considerable amount of work and present some new ideas, I think we must bear in mind that the classification of granulomata may remain as simple granulomata and epithelial granulomata. Burchard and Inglis quote half a dozen investigators who adhere to this classification, which is also based on numerous histological sections.

How shall we apply in our daily work the information brought out in Dr. Freeman's paper and Dr. Schroff's discussion. If it were true as some claim that all granulomata contain epithelial cells, which is denied by the works of Thoma, and Burchard and Inglis, it would mean that all teeth presenting granulomatous areas must be removed. My clinical findings do not accord with this. I have shown before this Section instances of granulomata which, treated without any destructive drugs or measures,

had disappeared. To explain this result is not easy. In order to prove that our explanation is correct, we would have to extract these teeth and make histological sections of the parts formerly occupied by the granuloma. Patients who have had a tooth saved in that manner would naturally refuse to have the tooth removed. We may assume that absorption of the granuloma occurs, and the area is probably filled in with new bone-tissue. This theory is not only suggested but strongly borne out by our radiographic findings.

The late Dr. M. L. Rhein claimed frequently and emphatically that he destroyed granulomata by incineration. He would carry the sodium-potassium compound on broaches into the granulomatous area and destroy it. The incineration of the granuloma produced, in his belief, pronounced irritation. Although I personally believe he was mistaken as to the cause, the granulomata did disappear. This he proved with his radiographs, and we know he was a clinician par excellence. I believe that when the cause that produces a granuloma is removed, the granuloma disappears. You may ask: Will it disappear in all instances? I am not prepared to say. We can't always get patients back for a check up. If we could get a number of such cases and watch them for a longer period of time, we could probably remove the doubt. I cannot subscribe to the removal of all teeth affected with granulomata, because I know that we have treated and cleared up a number of cases.

Harry Goldberg, D.D.S. (*New York City*): I have been associated with Dr. Freeman for some time, and have watched him do much of his work. He has been very conscientious in all his findings. The question has often been asked: Do not the bacteria in a granulomatous area diffuse through the cystic wall? Many persons have believed that, owing to the presence of this wall, bacteria do not work out into the blood stream. While I believe they do penetrate the wall, I should like to ask Dr. Freeman to discuss this question in his closing response.

Benjamin Horn, D.D.S. (*New York City*): I shall ask Dr. Freeman a few questions, and would like to have them answered from the point of view of pathology. What happens to a granuloma when it remains in place after an extraction? Has Dr. Freeman done any work on granulomata where teeth have been treated; if so, what is then the picture? I heartily agree with Dr. Schneer, and unfurl the banner of Pollia to "save teeth."

O. T. Rule, D.D.S. (*Stamford, Conn.*): I would like to ask Dr. Freeman whether his studies have reached a point where he can differentiate in

radiographs between virulent and non-virulent granulomata? Some years ago Dr. Rosenow believed that an x-ray picture having a dark area around the apex of a root showed where the battle had been taking place; that the battle had been won; and that this was rather a safer tooth to leave in than one in which the granuloma could be but faintly outlined, or was simply a slight departure from the normal in a picture of the surrounding bone. Such pictures show granulomata. Has anything definite been found to indicate which are virulent, and which are not? I presume that the size of the granuloma has nothing to do with the danger to the patient.

Dr. Freeman (*in conclusion*): This paper was just a preliminary survey. The collection of slides has been made over several years, and covers almost every phase of this condition at all ages. These cases were obtained from private practice, hospital, and clinic. All were treated and prepared in the same manner, so that we might have an unbiased picture. Some were from devitalized teeth, some from teeth that had degenerative pulps, some from teeth that had been improperly filled, and some from teeth with perfect fillings. In the next series, we are going to take individual cases, radiographs, blood picture, urine analysis, etc.—the entire systemic background, with the dental history of the extracted tooth and the histopathological picture. It may take five years; but if at the end of that time, we can arrive at a definite conclusion regarding this particular lesion, our efforts will not have been in vain.

Regarding Dr. Schroff's discussion, there is very little I can add, except that I do not want you to have the impression that we must remove a tooth in order to eliminate a granuloma. I do not do it in my own office, nor advocate it as a general thing. In view of the fact that it is absolutely impossible to differentiate clinically or radiographically between a simple granuloma and the epitheliated forms, I feel that some treatment other than root-canal therapy is indicated. We suggest either apicoectomy, or curettage of the soft tissue about the apices of these teeth after thorough root filling. This will prevent the possibility of any future development of a cyst.

I have seen Dr. Schneer's work and have the greatest respect for his ability. The series of cases that he showed recently were only roentgenographic studies. Do we know whether the tissues that fill-in these areas are really healthy tissues? They may appear so radiographically. They may even be bone lamellae. We must bear in mind that many scar tissues of regeneration are more or less infected. We do not know whether these areas are sterile or potential sources of infection. All of these problems

deal with focal infection. It is not merely a question of removing a tooth. We can extract the teeth of patients and yet their condition may not clear up. However, in a patient having a definite systemic condition that points to some focus, and the medico-dental history agrees with this indication, then we may feel justified in removing that focus.

Dr. Horn raised a question regarding retained granulomata. We did not do any work with these for the simple reason that, in curetting the sockets, we were liable to include some of the gingival epithelium. In a paper just published in the *Journal of the American Dental Association*, McCall discusses retained granulomata and residual infection.[4] I don't question the fact that some of a residual infection acts as a potent factor in a possible systemic infection, as with any other infection. As we have demonstrated with our slides showing a hematogenous communication, such residual granulomata or other areas, if left in place, would undoubtedly cause trouble.

Dr. Horn: Is not every granuloma fed by the tooth in position?

Dr. Freeman: Not necessarily. A patient having several devitalized teeth presented with an edentulous upper left area. Radiographs revealed a cyst about the size of a walnut. The history obtained from the dentist who referred the patient indicated he was certain this area was never that size. Previous radiographs had not revealed it. This residual area probably was an epitheliated granuloma, which had been left behind at the time of extraction. This however kept on developing of its own accord, without any tooth being in position and in any way influencing its growth. The cyst was removed from this edentulous area, where I am sure no tooth had been in place for at least five or six years.

The question of treated teeth was covered in our statement that in this series of 240 granulomata we took all types—any case that presented a so-called area. There is no question that several of these were treated teeth.

The differentiation of virulent and non-virulent granulomata has often been raised as a theoretical question. It simply brings back again the question as to whether the granuloma with its fibrous capsule is less capable of harming the individual than one in which there is merely a diffuse area. From actual clinical experience, checked by bacteriological findings, we find that the more virulent organisms are usually obtained from teeth that show the diffuse areas rather than from granulomata. We are very often

[4] See also McCall: *Journal of Dental Research*, 1930, x, pp. 113, 394.

justified in feeling that, where there is granulomatous formation, nature has combated this infection. As Dr. Schroff pointed out: in tuberculosis a typical tubercle formation shows nature's attempt to wall-off an infection. We do not like to classify these as granulomata, because the real substance of a granulomatous mass is not present. They are simply diffuse areas, probably showing definite reactions of the tissues to the infection, but not being able to confine it.

A RATIONAL TECHNIQUE FOR THE CONTROL OF CARIES AND SYSTEMIC PYORRHEA[1]

HAROLD F. HAWKINS, D.D.S.

Los Angeles, California

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I. INTRODUCTION

The control of dental decay by the proper manipulation of diet is an accomplished fact. Many investigators can testify that they have succeeded in causing the arrestment of active caries by fundamental changes in the diet: white lines around the necks of teeth have completely disappeared and open cavities have turned dark and over a period of years have shown, by the fact that they have not enlarged, that caries was completely arrested. There are two groups of factors in dental decay: on the one hand local factors, which consist of the bacteriological splitting of carbohydrate food with the production of lactic acid and the neutralization of that acid by the tooth itself; on the other hand, general or systemic factors that govern the composition of the saliva as to its concentration of calcium and alkaline salts, which have the power of neutralizing lactic acid. In the past the greatest effort to control caries has centered in an attack upon the local factors. The statement: "a clean tooth will not decay" illustrates the direction of this attack. That the toothbrush has been a helpful agent in reducing decay no one can successfully deny; that it will never hold caries within low limits is also very apparent.

[1] Read at a special meeting of the New York Academy of Dentistry, Yale Club, New York City, June 10, 1930. Some of the data were published in the *Proceedings of the Second Triennial Pacific Coast Dental Conference*, 1929, pp. 257–274.—[*Ed.*]

The use of specific germicides for the destruction of lactic-acid producing organisms, while helpful, cannot, I believe, control the situation. The mouth cannot be sterilized and rendered germ free any more readily than the skin. When we remember that bacteria under favorable conditions can double in numbers every fifteen or twenty minutes, we see how impossible such a control would be. Therefore, the major attack against caries should be made systemically and, if coupled with proper tooth brushing, the number of individuals showing decayed teeth should not exceed 5 percent of the population. This percentage, a conservative figure, can be reached by a reasonable amount of coöperation.

To make this paper complete, it is necessary to review material covered by former papers, with additions and changes made necessary by further research.

II. A REVIEW OF FORMER RESEARCHES

In studying over one hundred immune individuals it was very apparent that their immunity was not due to a technique in tooth brushing. On the whole less care was given than in other similar groups suffering considerable decay. A study of the composition of the saliva of the immune and carious showed that in the case of the immune to decay there was a highly alkaline saliva, or a high-calcium content and often both, while the saliva of those having extreme decay showed considerable acidity and a low-calcium content. (The saliva samples should not be taken within one hour following a meal or food of any sort. If this rule is observed and no violent exercise indulged in, the saliva is reasonably constant throughout the day.) It would seem logical, in the light of our findings, to state that the reason teeth do not decay when subjected to acids of fermentation is because of the presence in the saliva of alkaline salts and calcium, which neutralize the same. If it be true that those who are permanently immune to decay maintain in their saliva at all times a concentration of alkaline salts and calcium sufficient to neutralize the acids of fermentation, then it would seem logical that the permanently immune to decay could be used to establish a safe limit, based upon the alkalinity and calcium content of their saliva, beyond which caries could not result. We undertook to study this problem and have established a line that will

probably be slightly changed as more immunes are added to our tabulation, but we do not expect any marked deviation from its present position.

Fig. 1 relates to about one hundred and forty persons immune to decay. No one in this tabulation ever had a cavity of decay; it is a graph of strictly immune individuals. The reactions of the saliva were measured by a colorimetric hydrogen-ion test, which was selected in preference to an electrometric method because it was found possible to measure, accurately or roughly as the future will disclose, the actual buffer action of the saliva. By permitting the indicator to remain in contact with the salivary sample for about one hour, a slight or marked change took place in the reaction; this rise was always toward the alkaline, and at the close of this period usually reached an end point. We believe that the final reading at the end of an hour is very close to the true neutralizing value of the saliva. The neutral point in the hydrogen-ion scale is pH 7.0. Larger numbers indicate alkaline values, smaller numbers acid reactions. The calcium was registered by Kramer and Tisdall's method for blood calcium adapted to saliva. Every dot on the graph represents the reaction and calcium of an immune individual. It will be noted that most of these dots are on the alkaline side of neutral, which is shown by a vertical line. All dots on the acid side of neutral represent high-calcium values, some of which are quite acid. A diagonal line was established, which represents borderline cases, and can be used as a dividing line between immunity and decay. Several thousand specimens of saliva have been tested against this line, with the result that it seems to represent a fairly accurate demarcation between immunity and decay.

Fig. 2 shows the reaction and calcium content of the saliva of persons who, having active systemic pyorrhea, are immune to decay. By "active systemic pyorrhea" is meant the cases that show marked inflammation and hypertrophy of the gum tissue throughout the mouth, accompanied by pus production, general bone absorption, and often heavy salivary deposits. These cases are neutral, or fall to the acid side of our vertical line and to the right of our diagonal line. The so-called anemic form of pyorrhea, that is, where gum tissue does not show hypertrophy and inflammation but usually exhibits considerable tissue recession accompanied by bone absorption, falls to the alkaline

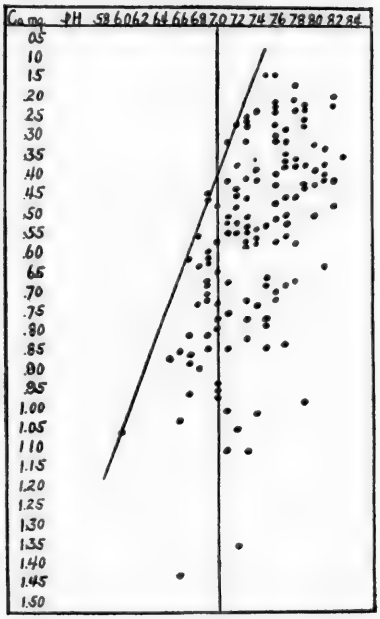

FIG. 1

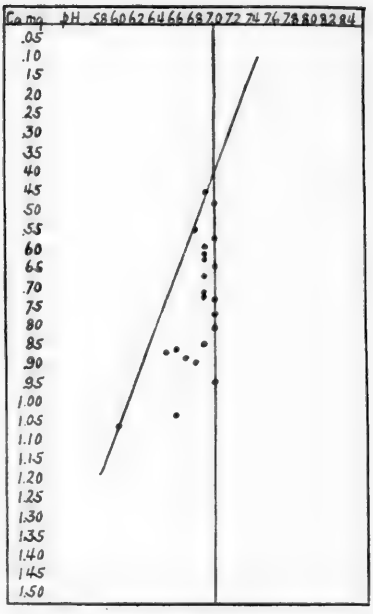

FIG. 2

FIG. 1. Immune to decay. The letters "Ca mg" at the top of the column of numerals, in each of the sixteen figures, refers to milligrams of calcium in 1 cc. of saliva.

FIG. 2. Immune to decay, with pyorrhea.

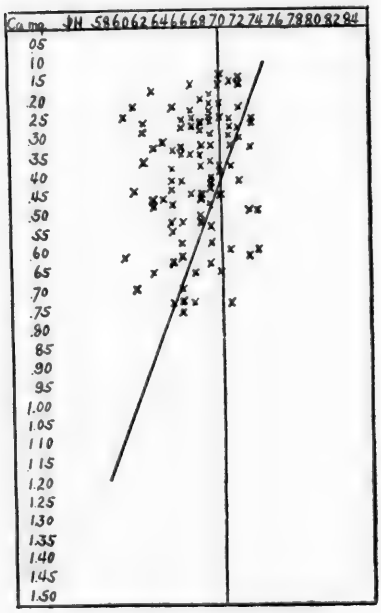

FIG. 3

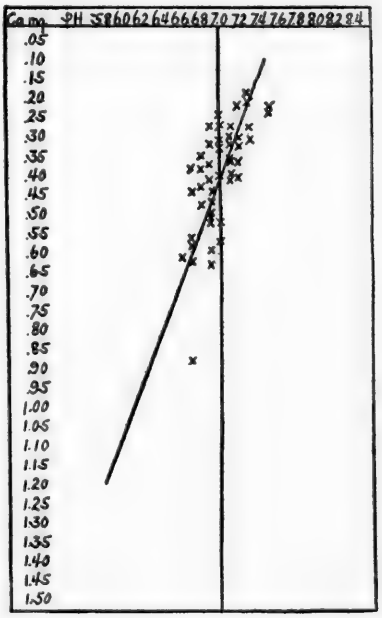

FIG. 4

FIG. 3. Active decay.

FIG. 4. Erosion and Class-V cavities.

side of the neutral line. The saliva of these cases shows a slight to marked reduction in the percentage of calcium. This type of pyorrhea we believe is progressing toward arrestment. There is probably such a condition as purely local pyorrhea, uncomplicated by systemic factors, caused by irregular teeth, open contacts, and faulty dental restorations. However, we note in many of these cases that the reactions are not ideal. While the local factors probably greatly outweigh the systemic, yet the systemic forces may contribute to the condition.

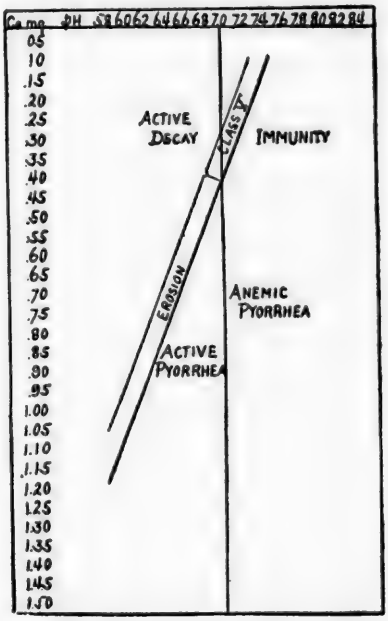

FIG. 5. Relationship of decay, erosion, and pyorrhea to immunity.

Fig. 3 shows the record of persons having active decay. All represented in this graph had many apparently active cavities. The big majority lie to the acid side of the diagonal line and represent low-calcium values. At the time of test a few were on the immune side of the diagonal line. Our records show that in those remaining on the immune side for any length of time, the cavities turned dark and developed what is known as "arrested caries."

Fig. 4 shows erosion and gingival decay, the latter known as Class-V cavities in Black's terminology. These cases tend to parallel the diagonal line. The saliva of these cases is not poor enough to permit active decay or good enough to secure immunity. Erosion cases lie to the acid side of the diagonal line and represent high-calcium values. These areas are always highly polished and often accompanied by considerable tissue recession. Gingival decay, or Class-V cases, are acid or slightly alkaline, with low calcium; the areas are not highly polished and there is no tissue recession. There is a close relationship between erosion and systemic pyorrhea, and an equally close relationship between gingival decay and the other forms of caries. There is no doubt that toothbrush habits and gritty dental pastes play an important part in removing decalcified enamel, with a resulting polished surface.

Fig. 5 is a graphic representation of the important facts in *figs. 1–4*. It shows areas of active decay, erosion, gingival decay, active pyorrhea, anemic pyorrhea, and immunity. The range of the reactions of the saliva, based upon several thousand tests, is from pH 6.0 to 8.4 (colorimetric). We performed a series of experiments on teeth subjected to a concentration of lactic acid of pH 6.0 for a period of two months without the slightest indication of etching of the enamel on drying. This would indicate that the saliva itself, no matter how acid, can never be a cause of enamel destruction. Mouth bacteria working on starch and sugars were able to develop concentrations of acid ranging from pH 4.5 to 3.3. Teeth subjected to lactic acid pH 4.3 for a period of two weeks lost so much of the intercementing substance that the loss could be readily seen on drying. This is conclusive proof that the acids of fermentation are of sufficient concentration to cause the destruction of tooth substance.

The density of the intercementing or interprismatic substance of the enamel varies considerably in different teeth. In mouths showing many active carious lesions, the intercementing substance is very much less dense than in teeth from immune mouths. This can be easily shown by taking a section of a tooth from a carious mouth and of one from an immune mouth, and then subjecting these sections to weak acid for a few days. If they are removed each day and allowed to dry, for observation, it will be seen that the etching is more rapid on the

enamel of the tooth from the carious mouth, and also that the etching is more intense at the surface of the enamel. On the enamel of the immune tooth the intensity of the etched surface is greatest at or near the dento-enamel junction. These conditions are the rule for many teeth.

The increased density near the surface or periphery of immune teeth, and the decreased density near the surface of carious teeth, are suggestive of a hardening or softening of the intercementing substance owing to conditions in the environment of the teeth. In the light of these tests it is very improbable that the pulp exercises any control over the density of the interprismatic substance of the enamel after eruption, notwithstanding the fact that other investigators have tended to show in the lower animals that the intercementing substance is permeable to dyes through the pulp.

To secure a better understanding of caries, erosion, and pyorrhea, it is necessary to ascertain the reaction and calcium content of the urine. The full-night specimen makes a desirable sample. Coördinated saliva and urine samples from the same individuals tend to show the following: as saliva becomes acid its calcium content increases, and as it becomes alkaline its calcium decreases. In the urine of the normal individual, and of those having systemic pyorrhea, the content of calcium fluctuates directly with the acidity. In caries, as the urine becomes more alkaline its calcium content increases, and as it becomes acid its calcium content decreases.

A very common condition causing, we believe, a serious metabolic disturbance, is an acid saliva accompanied by an alkaline urine. What are the factors that may cause urine to become alkaline and saliva simultaneously to become acid? We find the following are influential:

(1) An acute or pronounced chronic infection
(2) Pregnancy
(3) Consumption of a high-cereal or high-grain diet
(4) Regular use of magnesium salts

We find in a large percentage of cases, when the cereal or grain intake is extremely high, that the urine becomes alkaline and the saliva acid. We have found no other article of food, besides the cereal grains, that cause the urine to become more alkaline than the

saliva. In *Table 1* we show a few cases of urine more alkaline than the saliva, produced by infection, by pregnancy, by a high-cereal diet, and by a high-magnesium intake. In checking our recorded cases, we have found no natural immunes whose urines are more alkaline than their salivas. A similar observation on persons having active caries shows that the simultaneous occurrence of alkaline urine

TABLE 1

Reaction of the saliva and urine in relationship to infection, pregnancy, high-cereal diet, and magnesium intake

	pH VALUES	
	Saliva	Urine
Infection:		
E. H. Active pulmonary "T.B."	6.2	7.6
Eleven months on diet	7.5	5.6
F. D. S. Gall-bladder infection	7.1	8.3
J. P. Chronic abscess with sinus	6.7	7.1
A few weeks after removal of tooth	7.9	7.7*
Pregnancy:		
A. F. G. Five months from conception	7.1	8.0
Eight and one-half months from conception	6.6	8.0
Two months after birth of child	7.5	6.3
High cereal-diet:		
W. M. W.	6.0	8.1
Cereals removed for ten weeks	7.1	5.6
L. W. N.	6.7	6.7
Thirteen months on low cereal-diet	7.8	5.4
Magnesium salts:		
A. S. Constant use for one year	7.2	8.5
Three weeks after discontinuance	7.4	5.9
M. H.	6.8	7.8
Three weeks after discontinuance	6.9	6.0

* Not a night sample.

and acid saliva is very common, and the rule rather than otherwise; and with it goes a very heavy urinary calcium loss.

To restate briefly: the normal reaction of the saliva is alkaline; the normal reaction of the urine is acid. If for any reason these reactions become reversed, we believe a pathological condition then exists.

We find that in most individuals whose urines are more alkaline than their saliva, the reactions can be reversed by removing any

existing focal infection, and reducing the percentage of cereal products in the diet to a very low level. When we gain a better understanding of our problem in caries and pyorrhea, it can largely be reduced to the terms of biochemistry. Studies of total calcium and phosphorus in saliva, blood serum, and urine, have been quite illuminating, and make plain our observations. With very few exceptions we find that when the pH of a urine is alkaline the phosphorus is low; when quite acid, the phosphorus is high. In saliva the observations are the same. In a high-alkaline saliva we find a low phosphorus; in an acid saliva, a relatively high phosphorus. In *Table 2* we show our observations, rated from the average normal both in caries and systemic pyorrhea. In caries is found a low-blood and low-urinary phosphorus, and in the

TABLE 2

Relationship of calcium and phosphorus to caries and systemic pyorrhea

	Ca	P
Caries:		
Saliva	Low	High
Blood	High	Low
Urine	High	Low
Pyorrhea:		
Saliva	High	Low
Blood	Low	High
Urine	Low	High

saliva a relatively high phosphorus. Without an acid urine there would not be sufficient phosphorus intake to correct the low-blood phosphorus. The desirability of an acid urine has been previously emphasized. The blood calcium we note is high. This does not imply that the calcium intake is entirely adequate; we have every reason to believe that it is not. We must give special attention however to the phosphorus-containing foods, and develop an acid urine if we hope to control caries.

In systemic pyorrhea is found a very high urinary phosphorus and a relatively low salivary phosphorus, although the latter is above normal. The salivary phosphorus never drops to an extremely low level, hence the pH never indicates distinct alkalinity in active pyorrhea. The reaction approaches neutrality, as may be readily seen

from our graphs, and often reaches it; but it is believed at present it does not often pass to the alkaline side. For if that should happen, the tone of the tissues would go up owing to the alkaline reaction, and hypertrophy and inflammation would tend to disappear. The blood picture in pyorrhea shows that our task is to alkalinize and increase the calcium, as well as to restrict the phosphorus-containing foods.

In caries and pyorrhea the balance between calcium and phosphorus does not complete the picture. In checking two hundred immune individuals we found that the phosphorus content of the saliva did not

TABLE 3

*Comparative study of potassium, sodium, calcium, and phosphorus in common articles of food**

FOOD	K	Na	Ca	P
Milk, human	100	21.3	72.4	32.0
Milk, cow	100	35.7	84.0	65.1
Cheese, American	100	681.8	1047.4	768.4
Meat	100	24.9	3.4	63.7
Fish	100	22.3	6.5	68.8
Eggs	100	102.2	47.9	128.7
Flour, white	100	52.8	17.6	81.0
Flour, graham	100	8.1	8.5	79.7
Potatoes, white	100	4.9	3.3	13.5
Peas, fresh	100	4.6	9.8	44.6
Carrots	100	35.2	19.5	16.0
Spinach	100	16.2	8.7	8.8
Oranges	100	6.8	25.5	11.9
Apples	100	8.7	5.5	9.5

* Intended to show relationships uniformly per 100 parts of potassium.

vary with the pH. There was enough fluctuation in the results to take it beyond a reasonable chemical error, and the type of variance indicated clearly the presence of another factor. Phosphorus exists in the body in the form of mono- and dibasic phosphates. The monobasic are distinctly acid; the dibasic are distinctly alkaline. The evidence seems to indicate that the potassium content, notwithstanding it is low in blood serum, is of great importance in creating the dibasic phosphates. Here we deal with three factors: amount of calcium, amount of phosphorus, and percentage of potassium, in the forms of mono- and dibasic salts. The hydrogen-ion test can express the balance or amounts of mono- or dibasic phosphates in terms of pH.

Bunge has shown that when potassium is too high, in the body, sodium is driven out. The reverse also is true: a very high sodium intake causes the elimination of potassium from the body. The balance between potassium and sodium, and between calcium and phosphorus, are important factors. If we study Sherman's food tables as to the contents of potassium, sodium, calcium, and phosphorus, we will better understand why certain foods are favorable and others unfavorable in our study of caries. To facilitate ready comparisons we have multiplied the values for all of the foods by a factor that makes potassium 100 in each. In this way we can see at a glance whether potassium is relatively high or low. We suggest that human milk may be used as a standard for comparison. See *Table 3*. Other elements no doubt are factors in this chemical balance. The practical application of this knowledge, to individual cases, is shown in the case records below (*figs. 6–16*):

Fig. 6: W. M. W.—an individual with active decay living on a very high cereal-diet. Potatoes were substituted for cereal foods and more vegetables added. In the ten weeks covered by these tests, he passed from a condition of pronounced acidosis, accompanied by active decay, to a condition of slight alkalinity and immunity to caries.

Fig. 7. The reactions of a group of children placed them too close to our diagonal line for safety. A half-cup of orange juice and a small amount of cod-liver oil were fed each day for a month. No other change was made in the diet. At the end of this time the saliva was rechecked. The original tests are shown by x marks, the final tests by dots. A marked improvement in the alkalinity and percentage of calcium is indicated.

Fig. 8: R. W. T.—effect of a well-balanced diet on erosion covering a period of six months. The tests are numbered in the order in which they were made. The progress was not uniform owing to a failure at times to maintain the diet, but the final tests are very favorable and resulted in a good margin of immunity.

Fig. 9: L. W. N.—an individual on a high-cereal, low-vegetable diet, having active decay and pronounced nutritional anemia. Owing to poor appetite and low physical condition, the progress was slow. After seven months the patient's weight had greatly increased, and her color and general physical condition were exceptionally good.

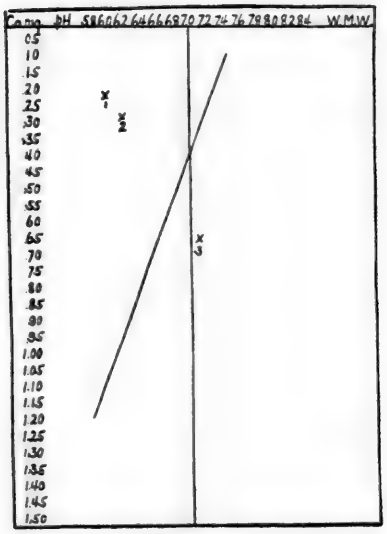

FIG. 6

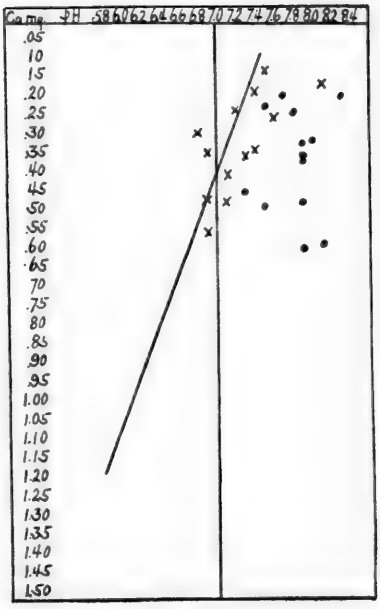

FIG. 7

FIG. 6. Effect of well balanced diet: active decay.
FIG. 7. Effect of orange juice and cod-liver oil: group of children.

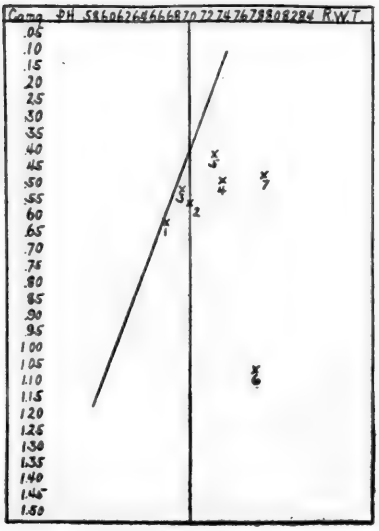

FIG. 8

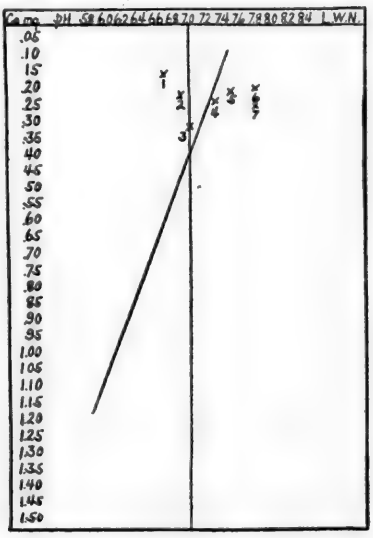

FIG. 9

FIG. 8. Effect of alkaline diet: erosion.
FIG. 9. Effect of well balanced diet: active decay.

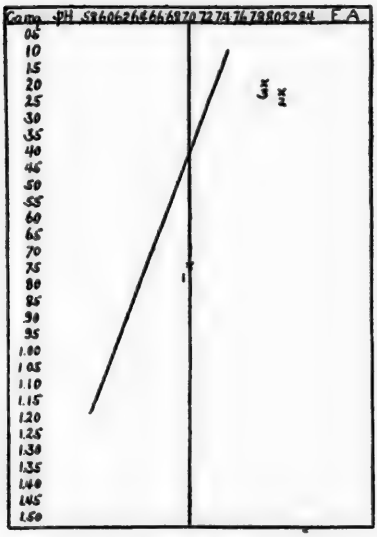

FIG. 10

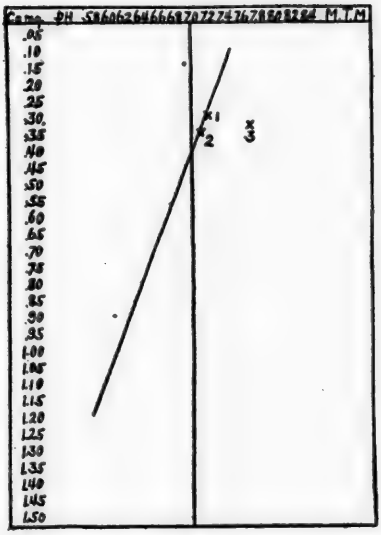

FIG. 11

FIG. 10. Effect of change of diet and the discontinuance of toxin absorption: pyorrhea.
FIG. 11. Effect of alkaline and low-residue diet: decay, with tonsil enlargement.

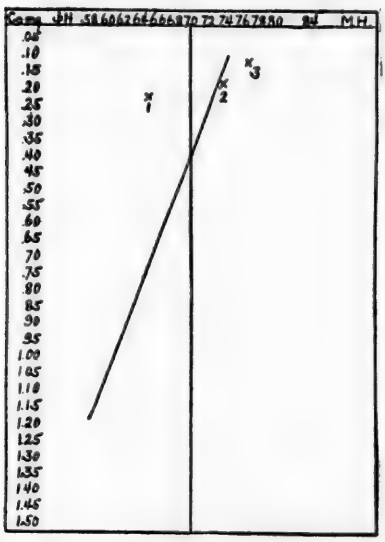

FIG. 12

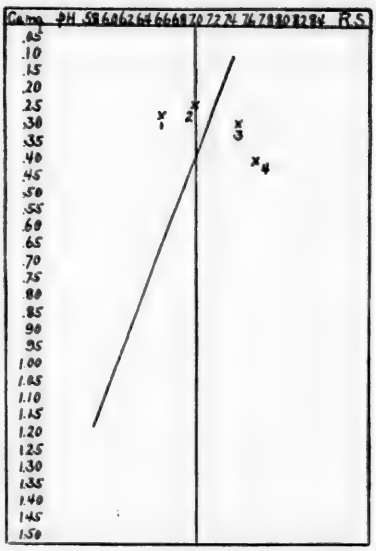

FIG. 13

FIG. 12. Effect of alkaline diet: decay.
FIG. 13. Effect of well balanced diet: decay.

Although the patient was safely immune to decay, several additional months of treatment would have been required to raise the calcium to the desired level.

Fig. 10: E. A.—a case of active systemic pyorrhea, with considerable bone absorption and pus discharge from many pockets. The patient was placed on a well balanced low-phosphorus diet having a decidedly alkaline potential. All the teeth from the upper arch, and a few from the lower, were extracted. In about eleven weeks the saliva was extremely alkaline, and there was a great reduction in calcium. His general health showed a marked improvement, which was probably due to the character of the diet and the discontinuance of toxin absorption.

Fig. 11: M. T. M.—a six-year old boy on a high-cereal, high-vegetable diet, had a great many fillings in the first teeth, and two cavities in six-year molars. One tonsil was greatly enlarged, and the patient was not in good health. No progress was made in this case until the tonsil was reduced to fully half its original size by the low food-residue in the prescribed diet. In about thirteen weeks on a well balanced diet, the saliva was sufficiently alkaline, with a slight increase in calcium. The patient's general health was much improved.

Fig. 12: M. H.—a sixteen-year old girl suffering from active decay with many Class-V cavities. In two months on a well balanced diet the tests showed that she was immune to decay. High-vitamin D and phosphorus feeding, accompanied by an adequate amount of calcium, would have been necessary to bring the salivary concentration of calcium to a higher level.

Fig. 13: R. S.—a woman about thirty-eight years of age, with considerable decay. During a period of eight months she passed to a safe margin of immunity. The progress was delayed by a severe attack of influenza.

Fig. 14: L. R. P.—a boy about eighteen years old, with considerable decay, especially along the gingivae. In four months he was immune to decay owing to favorable diet. At the indicated rate of progress two more months would have been required to give an ideal margin of safety.

Fig. 15: E. H.—an active case of tuberculosis, with considerable decay. The tests covered a period of eleven months, at the end of

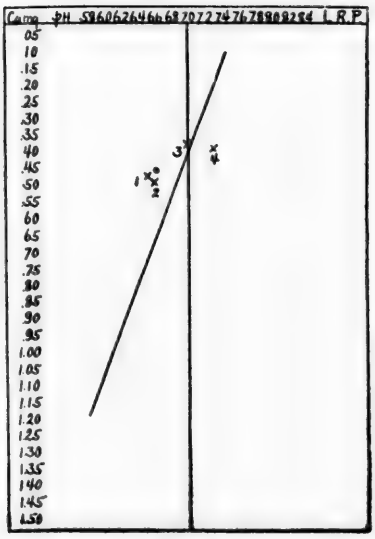

FIG. 14

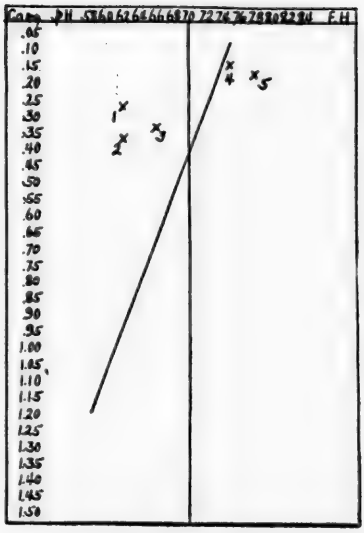

FIG. 15

FIG. 14. Effect of alkaline diet: gingival decay.
FIG. 15. Effect of well balanced diet: active tuberculosis, with considerable caries.

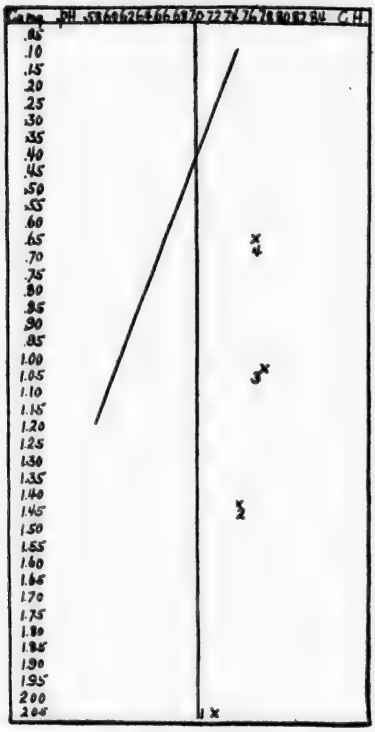

FIG. 16. Reduction of salivary calcium by suitable diet.

which she was making rapid strides toward complete recovery. Several additional months would have been required to give her a safe margin of immunity, so far as caries was concerned.

Fig. 16: C. H.—a sixteen-year old girl having extremely heavy salivary deposits. The deposits were so heavy that only the biting surfaces of the lower arch could be seen. Chisel and mallet were used to remove the accumulation. A highly alkaline diet was recommended, sufficient but not excessive in calcium. The phosphates were reduced. In five months the calcium content of the saliva was less than one-third the original amount, and she was able to keep her teeth reasonably free from salivary deposits.

Many other cases might be shown to illustrate the possibilities of improvement in alkalinity and calcium content on a suitable diet.

III. A PRACTICAL APPLICATION OF THE RESULTS OF FORMER RESEARCHES TO CHILDREN IN THE LOS ANGELES ORPHAN HOME

A practical application of the foregoing systemic factors for the control of caries was undertaken at the Los Angeles Orphan Home in October, 1927, and completed in January, 1929. The reaction and calcium content of the saliva, of one hundred children from three to thirteen years of age, were determined. Bite-wing x-ray films were made of each case and the number of open cavities, fillings, and mouth conditions, thoroughly examined and recorded. A research committee of three from the Los Angeles County Dental Society, headed by Dr. Walter G. Crandall and the Supervising Dentist of the Los Angeles County Health Department, Dr. Hazel Merrick, made a careful oral examination of every mouth and all data were carefully recorded. It was found that the average incidence of caries was over 5.5 cavities and slightly over 2 fillings per child. The mouth hygiene was very poor, and a great many children had hypertrophy and inflammation of the gum tissue. No great effort was made to stimulate tooth brushing, as the test was to be a check on the systemic factors involved, not on mouth hygiene.

The diet of the children was changed. A moderate-meat, low-cereal, high-alkaline and high-calcium diet was fed during the period of the test. Frequent tests of the salivary reaction and calcium content were made. When the tests showed that a child was low in alkalinity

or calcium, the diet was supplemented with orange juice or cod-liver oil. During the period of the test about 25 percent of the children left the institution, most of them during the summer vacation, and on these it was not possible to make a final check. On the remaining seventy-four children, another set of bite-wing films was made and all of these children were finally "checked out" by the same examiners. No new cavities were found. The gum tissue in the mouths of the children was good, all evidence of inflammation and hypertrophy having disappeared. The mouths were not as clean as when originally "checked in," and there was a slight increase in the percentage of tartar. The result of this test or demonstration showed that the systemic factors responsible for caries and gum tone were completely under control. The fact that there had been 555 cavities and filled teeth in the group indicated a very marked tendency to caries. That this tendency was completely checked is very evident from the result obtained.

In January, 1930, another check was made on the remaining forty-one children of the original group. Bite-wing films were made, and the same personnel conducted the checking. One cavity was found under a Class-II filling, which had a large overhang permitting constant food retention. This was the only cavity that developed at the institution in the two and one-third years of the demonstration. From the daily-diet records for the two years, it may be clearly seen that the percentage of grain or cereal foods for 1929 was slightly in excess of that for 1928. This fact may account for the failure in this one case, notwithstanding the favorable conditions for caries. If the diet is sufficiently low in the total amount of grain foods, I believe there is no reason for the development of cavities in most structural defects.

While the chemical balance of a diet is all important, we must not forget that the physical character of the food also plays a part. Such cereal foods as wheat, which carry a high-gluten content, hold the fermenting starch in close contact with the teeth and increase the tendency to caries. Our research shows that gluten is not physically changed within the range of the reactions of the saliva. Not until we reach an acidity of about pH 3.0 does disintegration take place.

The vitamin content of food is important, and in the case of vitamin D bears directly upon our problem. Vitamin D increases the salivary

calcium, an indication that it induces a lowering of the blood calcium, and corrects the faulty calcium and phosphorus balance in caries and rickets. Our research at present tends to show that vitamin D acts directly on phosphorus, increasing its absorption into the body, and acts indirectly on calcium. The latter varies inversely with the blood phosphorus. Alkaline urines become acid, and show a marked increase in phosphorus, in cases of caries when the patients are fed viosterol. While it may be true that, without vitamin D, normal individuals, fed a diet balanced as to calcium and phosphorus in the ratio found in human milk and in bones, can maintain a normal balance between calcium and phosphorus, yet our observations seem to indicate that individuals for a considerable length of time on cereals have their capacity for the normal absorption of phosphorus reduced, and require sunbaths or high-vitamin D feeding to re-establish such capacity. A study of common foods in *Table 3* shows that of the foods there listed, orange is the only one having a "correct" calcium-phosphorus balance. A very few others, such as cauliflower, celery, and lemon juice, have this balance. We also note that the other foods listed have an excess of phosphorus. Yet we see in caries a phosphorus deficiency. It seems evident that the body can not utilize the available phosphorus without the aid of vitamin D. In the practical control of caries, vitamin D is essential, but in the control of systemic pyorrhea the intake of this vitamin should be restricted.

There has been considerable research by other workers in regard to the value of vitamin C in caries and pyorrhea. We have been unable to confirm this finding. Vitamin C has not yet been isolated as a chemical entity. In most of the experiments, orange juice has been used as a source of vitamin C. Orange juice has fairly high contents of calcium and dibasic phosphates, and is used often to correct acidosis of the acid-ash type. When we feed orange juice as a source of vitamin C, we produce profound changes in the body as to calcium and phosphorus. In the face of these facts we cannot attribute all the favorable influences of orange-juice feeding to its vitamin-C content. Our experiments lead us to believe that the dibasic-phosphate content is the chief factor in the improvement in gum-tone for, after feeding orange juice, the saliva becomes distinctly alkaline. Inorganic salts by mouth, or hypodermically administered, will, if the saliva is made

distinctly alkaline, cause a marked improvement in mucous-membrane tone. Additional experiments seem to be indicated to establish the exact value of vitamin C in both caries and pyorrhea.

IV. SUMMARY OF GENERAL CONCLUSIONS

Caries exhibits in the blood stream a faulty balance between calcium and phosphorus, in which there is a high blood-calcium and a low blood-phosphorus. The phosphates are not only low but deficient in dibasic forms, a condition that is reflected in the saliva by an acid reaction and low calcium, and in the urine by an alkaline reaction and a high calcium.

Systemic pyorrhea also exhibits a faulty balance between calcium and phosphorus. The blood picture is a low calcium and a high phosphorus, just the reverse of caries. The excessive phosphates are usually deficient in dibasic salts but not to so marked a degree as in caries. This condition is usually reflected in the saliva by a moderately acid to neutral reaction, the calcium being excessive. In the urine the reaction is very acid, and the calcium loss is relatively low.

The attack on these problems must be made systemically through diet. Gratifying results have been demonstrated in group and in individual cases. High dibasic-phosphate and vitamin-D feeding in caries, and high-calcium feeding in pyorrhea, coupled with more dibasic and decidedly less monobasic phosphates, are essential.

The technique of control herein outlined is based entirely on biochemistry. It has been shown that the faulty balance can be ascertained, the indicated corrective diet instituted, and the progress of the case followed graphically. This method of control is a satisfactory and rational method of preventing and controlling both dental caries and systemic pyorrhea.

[After Dr. Hawkins finished reading his paper, and before its discussion was begun, Dr. Charles F. Bödecker presented a paper entitled "A new theory of the cause of dental caries." This paper has not been presented for publication. It was in substance a repetition of the paper published by Dr. Bödecker a year earlier: *Dental Cosmos*, 1929, lxxi, p. 586.—*Ed.*]

V. DISCUSSION

William J. Gies, Ph.D., Sc.D., LL.D., F.A.A.P., F.A.C.D. (New York City): Shortly after I received the invitation to discuss Dr. Hawkins' paper, I was favored with a printed copy of it. The President sent it without comment, except to say that it would help me in preparing my discussion. I took the hint and assumed that he meant I was expected to come to this meeting with a carefully prepared and thoughtful analysis of the speaker's paper. As the Chairman has said, I have been interested so long in the promotion of dental research, and in advancing its interests among dentists, that it is a deeply gratifying experience, as the years pass, to find dentists attacking the problems of dental science with increasing understanding, and as a group succeeding more and more in the solution of dental problems. Consequently, with this feeling I naturally listen to discussions of dental research with great sympathy and concern, and never find it in any sense pleasant to disagree with any who have conducted it. Dr. Hawkins represents a group of relatively young dentists who are giving increasing attention to dental research. If in disagreeing with some of his explanations I might by any possibility discourage him in his further efforts, I should be very unhappy. One who undertakes research, like one who proceeds with any constructive effort, must ordinarily expect to find himself frequently in places of great uncertainty, where several explanations, or several conclusions, are logical alternatives. Of course, in receiving and discussing scientific work, we must at all times be reasonable but also actively skeptical. I shall now read the discussion that was prepared with special concern, in accordance with the dignity of the occasion and in full justice to Dr. Hawkins.

The essential feature of the paper is the statement that Dr. Hawkins and his colleagues at the Los Angeles Orphan Home observed that, by a change of food to "a moderate-meat, low-cereal, high-alkaline and high-calcium diet," supplemented in some cases with orange juice or cod-liver oil, seventy-four children, from three to thirteen years of age, who, at the beginning of the observations, had an average of 5.5 cavities and slightly over 2 fillings, per child, continued through a period of fifteen months without acquiring any new cavities. This was found regardless of the fact that "no great effort was made to stimulate tooth brushing, as the test was to be a check on the systemic factors involved, not on mouth hygiene," perfect prevention having been noted throughout the fifteen months, although at the end "the mouths were not as clean as when originally examined." Then a year later, emphasizing this happy outcome, only one new cavity was found in forty-one of the children who remained in the school under the same condi-

tions. The new cavity developed beneath a "filling with a large overhang permitting constant food retention." This, I believe, is the most important practical matter that Dr. Hawkins presented tonight, all of the introduction having led up to this general conclusion.

If this important result can be obtained consistently by the same means, by *other* observers and on *other* children, Dr. Hawkins will have discovered one way not only to arrest the progress of dental caries, but also to prevent it. Until such repetitions and confirmations are reported, however, Dr. Hawkins' findings should be received, of course, with suitable doubt and reserve. The history of science, of medicine, and of dentistry is largely a record of misunderstandings of the conditions, and of misconceptions of the significance, of experimental and clinical observations.

Dr. Hawkins ascribes his results to conditions of diet that include *low* content of cereal, *medium* content of protein, *high* content of calcium, and *high* potential alkalinity. The following quotation from the issue of the *Journal of Dental Research* now in press—comment that emphasizes the importance of diet—will interest you not only as directly disagreeing with Dr. Hawkins' *explanations* of his results, but also as indicating the need for further clarification of the recorded observations in this important field:

"The hygienic condition of certain of the inhabitants of the Island of Lewis in the Hebrides could scarcely be worse. They live in 'black houses,' the walls of which are often as much as five feet thick. There is no chimney and only one opening in the house. Peat fires are kept constantly burning. Domestic animals occupy the building with the family. Chickens roost on the beds, tables, and chairs. The children are rarely ever taken out of the house until they can walk, yet rickets is almost unknown on the island; and, according to the Report, 'the most striking fact in the adult population is their beautiful teeth—a testimony to the absence of rickets in infancy.' Furthermore, the infant death-rate is among the lowest in the British Isles. . . . . The staple articles of food are fish, oatmeal, and eggs. Milk is scarce and vegetables almost unknown. Fish liver is a favorite dish. Such a diet is high in protein and phosphorus, and low in calcium and potassium. It has a relatively high potential acidity and is probably also rich in vitamins. Such a combination of dietary factors apparently is able to overcome the deleterious effects of poor hygiene, filth, and lack of ultraviolet radiation."[2]

The four dietary factors at the Los Angeles Orphan Home to which Dr. Hawkins ascribes special preventive virtue were *moderate* protein, *low* cereal, *high* calcium, and high potential *alkalinity*. But the diet common among the people on the Island of Lewis, as described in the foregoing

[2] Jones, Martha A.: *Journal of Dental Research*, 1930, x, p. 301. The data were compiled from a "Report on the physical welfare of mothers and children: Scotland," Vol. III, 1917. Published by the Carnegie United Kingdom Trust, Dunfermline, Scotland.

quotation, disagrees in quality with each of Dr. Hawkins' chemical criteria; that is to say, it is *high* in protein, *high* in cereal, *low* in calcium, and high in potential *acidity*. The conditions in the Island of Lewis to which the foregoing quotation alludes have been operative there for many years, and the quoted observations refer to a larger number of people through a longer period of time. In these two comparative instances, two sets of dietary factors, representing opposites in nutritional quality, are reported as having had exceptionally beneficial effects on teeth. This striking disagreement in scientific *explanations* justifies the opinion that errors have been made in arriving at the reported conclusions, in one case or the other, or both; or that essential factors, which were overlooked or disregarded in the observations, have had important favoring influences.

Dr. Hawkins does not indicate certain important types of detail regarding the general condition and environment of the children at the beginning of his observations, such as the nature of the diet they had been receiving and from which they changed at his direction, the ingredients of the diet to which he ascribes his results, etc., although his comment about their teeth and mouths, at the start, suggests that they were far from normal. Whether his new diet corrected previous gross nutritional deficiencies cannot be determined directly from his paper. Regarding adjustments to his experimental procedure he says: "When the [salivary] tests showed that a child was low in alkalinity or calcium, the diet was supplemented with orange juice or cod-liver oil." How much of each was given, and exactly why, is not indicated, but in the case of cod-liver oil presumably to correct an assumed vitamin deficiency. Many who ascribe extraordinary nutritional potency to vitamins, and urge their free *addition* to diet, seem to forget that every food may be poisonous when taken in excess, as determined by its own qualities. Evidence is rapidly accumulating that excesses of vitamins or their carriers, or both, may be very harmful. The last important paper I have read on this subject, which appeared a few days ago, described serious heart lesions caused by cod-liver oil,[3] which, in doses smaller than those commonly given to children for long periods, may induce morphological alterations of a degenerative nature throughout the whole mass of the heart, and consequent perturbations in its functions. Summer sunlight had a counteracting influence, but vitamins B and C in marmite and lemon juice, respectively, did not exert protective influence.

In passing I quote another view of the uncertainties in current interpretations of data on dental nutrition, especially on the influence of sunlight,

[3] Agduhr and Stenström: *Acta Pediatrica*, 1930, ix, pp. 229–391.

usefulness of milk, etc.: "In Holland . . . . one sees a hardy sunburnt peasantry. Their cows fill every meadow, and yet the teeth of these peasants seem to be in a worse state than the teeth of people in our own [English] factory towns."[4]

Please note that I am emphasizing the importance of correct explanations and interpretations. I would not wish to suggest that Dr. Hawkins did not find what he says he found. I would not wish to suggest to any observer that he did not see what he saw. But his statements about the significance of his findings or observations are suitable matters for discussion. Sometimes the investigator thinks he is looking north, when in reality he is looking south; and even a compass may get out of order and be misleading.

Regarding the use of orange juice in his experiments, Dr. Hawkins appropriately alludes to associated factors in the juice that might be more influential and significant than the vitamins therein. But he also emphasizes the current lack of clear interpretations in this field when he says: "There has been considerable research by other workers in regard to the value of vitamin C in caries and pyorrhea. We have been unable to confirm this finding." He adds, "We cannot attribute all the favorable influence of orange-juice feeding to its vitamin-C content . . . . and additional experiments seem to be indicated." In the same spirit, I suggest that we cannot ascribe to diet alone all the favorable results reported by Dr. Hawkins, and that additional experiments, for verification or correction as the case may be, are indicated.

In Dr. Hawkins' paper there is much expression of belief. In a description of research, statements of belief are indirect confessions that facts are lacking. Thus on page 217 Dr. Hawkins said: "If the diet is sufficiently low in the total amount of grain foods, I believe there is no reason for the development of cavities in most structural defects." Yet, in the paper, there is no evidence that pits and fissures were given special attention, and no basis of fact to justify this belief.

I have already said more than enough, in general comment, to indicate that in evaluating the significance of Dr. Hawkins' work, a reasonable skepticism forces itself into the foreground of a balanced discussion. But a few allusions to several biochemical details, of many that might be discussed, may not be amiss.

Dr. Hawkins refers to salivary composition as a criterion of dental conditions, but it is not clear whether he concludes that *all* of the various hypothetical dietary preventive influences were registered upon the teeth

[4] Morris: *Dental Magazine and Oral Topics*, 1930, xlvii, p. 462.

from *within* their structure, or *externally* from the saliva, or by both means. That he believes adequate diet may be preventive of caries, by giving protective power to saliva, seems to be indicated by such statements as this in his paper: "It would seem logical, in the light of our findings, to state that the reason teeth do not decay [in cases of immunity] when subjected to acids of fermentation is because of the presence in the saliva of alkaline salts and calcium, which neutralize the same." This quotation presents some biochemical anomalies. It has never been shown that a tooth in a case of immunity would not be denuded of its enamel and then decayed, at any given locus, if fermentable material were repeatedly lodged and recurrently renewed at that place on the enamel, and if, at the same time, no substance inhibitive of bacterial action at that point were present? The quoted statement also assumes without warrant that saliva bathes all tooth surfaces, including pits and fissures, the inside of mucin plaques, the interior of retained food masses, etc., sufficiently to neutralize immediately all of the acid that might be formed on any dental surface under any condition of oral fermentation. Dr. Hawkins explains the occurrence of one cavity in the second year of observation by saying it was due to fermentation of retained food at the edge of an imperfect filling. But, if his interpretation is correct, why did not the alkaline saliva act in this case to prevent decay?

In the paper presented by Dr. Bödecker the view was expressed that the protective influence against decay is normally exerted *from within the tooth* by something that arrives on the enamel surface at an adequate rate and in an amount sufficient *immediately* to neutralize all acid that may there be destructively produced. Later I shall suggest a very simple way to test Dr. Bödecker's theory (page 227).

The reaction of the saliva, upon which Dr. Hawkins bases some of his guiding principles, was determined by a procedure involving deliberate delay after its collection. Important ordinary variables in the secretion and collection of saliva are not mentioned, but Dr. Hawkins made this statement: "By permitting the indicator to remain in contact with the salivary sample for about one hour, a slight or marked change took place in the reaction; this rise was *always* toward the alkaline, and at the close of this period *usually* reached an end point. We believe that the final reading at the end of an hour is very close to the true neutralizing value of the saliva." (Italic not in the original.) I regret to have to say that this was a faulty procedure. The "true neutralizing value of the saliva," at any time *physiologically*, so far as the prevention of dental decay is concerned, is the power exercised in the mouth *only*. Carbon dioxide rapidly escapes from saliva

by dissociation of contained bicarbonate, thus tending to increase the basicity of the mixture. Bacteria rapidly develop in saliva, tending often to produce basic matter in heavy preponderance over acidic. At one place in the paper Dr. Hawkins, calling attention to the rapid evolution of bacteria wrote: " . . . . . bacteria . . . . . under favorable conditions can double in numbers every fifteen or twenty minutes." He seems to have overlooked their ability to keep on growing in the saliva, after its collection, and to keep on changing its reaction. He says, to repeat, that, during the wait for the collected saliva to acquire its "true neutralizing value," the reaction rose and that this rise was *always* toward alkaline—that is to say, the saliva *always* became more alkaline (less acid) outside of the mouth than it was inside of the mouth. But the only important power of the saliva for the prevention of dental caries is that exerted *within* the mouth, and at specific locations on the teeth. These departures from a biological basis for comparison give to the data, and the graphs for reaction, a synthetic and not a natural significance.

I am reminded that fifteen years ago we heard a good deal about a similar salivary alkalinity as a factor in preventing dental decay. A corresponding numerical coefficient was proposed as a useful criterion in diagnosis and control. This coefficient represented much earnest work similar to that shown by the "dividing lines" in Dr. Hawkins' graphs. After several years of debate, in which I participated skeptically in some papers in the *Journal of the Allied Dental Societies*, the doubt was found to be completely justified and the subject has become a part of forgotten lore.

If Dr. Hawkins alluded to calcium as a dietary factor that operates against caries from *within* the tooth, rather than by way of the *saliva*, to affect the quality of enamel, then he must assume or have learned that calcium does so by passing from the *blood* into the enamel, and there entering reactions having anti-carious effects. If he assumes, further, that such anti-carious effects are brought about by *nutritional* changes *within* the *enamel*, then the following conditions make such assumptions or theories at present untenable, pending discovery of a new condition that would overrule all of them:

(1) Enamel contains neither cell nuclei nor cell enzymes, which are essential factors in local nutrition as distinct from exchange of materials by diffusion only.

(2) The permeability of enamel for salts and gases is so slight in degree that in a recent investigation, which included the greatest refinements of accuracy, relatively long periods were required, under favorable conditions, to effect penetration.

(3) There are no *soluble* (serum, lymph) *proteins* in enamel. Recent investigations have also demonstrated that nucleoprotein and glycoprotein are completely absent, showing that proteins and lymph containing them *do not traverse the enamel* either from *within* the teeth or from *without.*

(4) The only protein in enamel—the substance of the organic matrix—is a form of keratin that resembles the chief protein in ectodermal structures in general. The proportion of keratin in enamel is less than 0.5 percent, and may not be more than half this very slight quantity. Enamel keratin, which is insoluble in lymph and chemically inert, has no apparent relation to any *nutritional* change in fully formed enamel, although its presence in the enamel of erupted teeth, presumably as a residuum, seems to increase the elasticity of the enamel and to reduce its brittleness.

This summary ends my prepared discussion. The summary was intended to show that, if Dr. Hawkins believes decay in his children was prevented by the responsive passage of calcium or anything else from *within* the tooth to the enamel *surface*, there to serve as a protection, such an opinion is not yet tenable. It happens that this summary applies also to the theory stated in Dr. Bödecker's paper, to the effect that caries is normally prevented by "lymph" that passes from the inside of the tooth to the outside, there instantly neutralizing destructive acid and thus completely protecting the enamel surface from corrosion by the acid. An objective discussion of this conception of protection, applicable alike to Dr. Hawkins' work and to Dr. Bödecker's theory, may not be amiss on this occasion.

Dr. Bödecker, in his paper, alluded to the fact that some years ago I volunteered to endeavor to ascertain whether what he called an "organic matrix" in enamel was in reality "organic," i.e., albuminous.[5] He had used a celloidin-acid decalcification process to isolate material that he believed was albuminous in quality, but during a period of about twenty years this material had not been subjected to a crucial test designed either to establish its real nature, or to remove reasonable doubts regarding it. His experiments had not shown that the "organic matrix" was not derived from the celloidin in whole or in part by a process the reverse of petrefaction. He will remember that while, under these circumstances, I frankly expressed doubts regarding the chemical quality of the matrix, I also indicated a way to eliminate the doubts—and then removed them by a simple and direct test, thereby converting a disputed opinion into an accepted demonstration.[6] In the same spirit, and for an impersonal scientific purpose, reminding Dr.

[5] Bödecker: *Journal of Dental Research*, 1926, vi, p. 130.
[6] Gies: *Ibid.*, 1926, vi, p. 143.

Bödecker of a discussion we had in March, 1929, on this subject, I again suggest a very simple, direct means of testing his theory, so that instead of long and fruitless discussions without any direct confirmation, the doubts, as in the case of the "organic matrix," can be promptly removed and the truth established. The simple and direct test to which I refer might be conducted as follows: Find a person who meets all of Dr. Bödecker's criteria of perfect health—in whom the so-called enamel "lymph" would, to satisfy his theory, be hypothetically most quickly responsive protectively. Then, with that person's consent, determine—on a selected small locus on a sound tooth—the action of a common oral-fermentation mixture of typical acidity. In this effort, the presence of the acid mixture upon the tooth, and other conditions, would be carefully controlled by suitable means as to definite and restricted location on the surface, duration of contact, undisturbed action, etc.—in some tests to determine, as with a "loopful" of the acid mixture, merely whether the enamel surface would be corroded; in others, as with short narrow tubes hold vertically in place, whether the enamel surface would be damaged, *and also whether the acidity of the applied liquid would be wholly neutralized, or diminished in degree.* If, with all variables under control, the enamel were not affected, but the acidity were diminished, the theory that in "healthy" persons a responsive enamel "lymph" instantly moves to the point of attack, and there prevents decay by promptly neutralizing hostile acid before it can react with the enamel to any extent, would have a foundation in fact. And elaborations of the direct test here proposed would provide desirable additional information. Thus, such "elaborations" would be applied, for example, to pits and fissures, where the distance that the hypothetically protective "lymph" must travel through enamel *to meet the destructive acid* is the *shortest.* These "elaborations" would doubtless then show why *decay is most prone to occur* where this "distance" is the *shortest* and where, consequently, the conditions are most favorable for the display, by the so-called "lymph," of the hypothetically protective action ascribed to it in Dr. Bödecker's theory.

Originally when I suggested to Dr. Bödecker that doubts would be eliminated by applying the biuret test to particles of his "organic matrix," to ascertain whether it was truly organic—which I did in his presence—the positive results obtained in a few minutes at the first trial, and repeatedly afterward, decisively confirmed his opinion. I again suggest that a simple test like the one just outlined would be similarly conclusive in removing doubt regarding his theory of normal protection against decay by the action of a hypothetically responsive enamel "lymph." If such an experiment should show that the acidity of a typical oral-fermentation mixture is diminished,

and that simultaneously the *enamel is attacked*, there would be nothing left of the theory. If, on the other hand, the acidity were diminished but the *enamel remained undamaged*, the theory would be confirmed at the point where at present the evidence is against it.

Charles F. Bödecker, D.D.S., F.A.C.D. (New York City): You can well imagine that the work of Dr. Hawkins has pleased me considerably, because it suggests that there is a close metabolic connection between the teeth and the body as a whole. I am sorry that I cannot discuss it from the biochemical standpoint, but that has already been done well by Dr. Gies. Dr. Hawkins has had somewhat the advantage of other investigators of saliva, in having all his results point definitely in one direction. That, to me, is unexplainable because twenty years ago, as Dr. Gies has already pointed out, intensive researches were conducted on the saliva and we did not gain any such one-sided picture. If it turns out to be as Dr. Hawkins says, the problem is going to be definitely simplified. A number of investigators have suspected that dental caries and periodontoclasia are really symptoms of one condition at opposite extremes. We clinicians very often note that when dental caries is rampant, periodontoclasia is not present, and vice versa; and even that in the same individual conditions change; that is, if decay disappears, periodontoclasia may set in.

Dr. Hawkins' clinical observations of individuals in homes, in connection with the possibility of arresting dental caries, is interesting. At Columbia, a committee for the investigation of dental caries has developed an instrument that you may be interested in seeing. It will be exhibited if the President invites Dr. McBeath to indicate the manner of testing the exact depth of a cavity by means of an electrical apparatus (see page 230).

Dr. Hawkins' observations are undoubtedly a heavy indictment of cereals. There is no doubt that a number of generations have been brought up on a high-cereal diet and dental decay is rampant. On the other hand Dr. Gies has indicated instances in which the cereal elements were exceedingly high in the diet, and yet dental decay did not take place. I believe there is one factor which may throw light on this paradox, and that is that the conditions Dr. Gies cites related to individuals living in extreme poverty. I believe that the average person suffers from overeating, while those that Dr. Gies cites certainly did not overeat; in that way possibly the conditions were somewhat bettered. Dr. Gies has already touched on saliva and food stagnation. The saliva does not reach all parts of the mouth equally, particularly not under the mucin plaques. It is for this reason that the outside as well as the inside of the tooth, is affected differently in its

reaction to dental caries. I believe that the saliva normally has a beneficial influence. Dr. Hawkins brought out the fact that caries-immune and caries-susceptible teeth react differently when subjected to acid. Does that not suggest that there is a difference in the enamel? May it be the manner of crystallization of the mineral salts in the enamel? Clinically as well as experimentally there is a difference in caries-immune and caries-susceptible teeth.

Dr. Gies some time ago made some experiments with trypan blue by feeding this dye to young dogs before the teeth were formed. This trypan blue was deposited in the later developed teeth and apparently also in the enamel. Dr. Gies formed the opinion that as this dye remained in the enamel, there is no possible passage of a current of fluid through the enamel, for the dye would be thus washed away.[7] I have been told that the molecule of trypan blue is complex and large, and I draw the following simile which we see going on here in New York frequently in building our large houses with their enormous heating plants. Have you ever noticed that the large boiler is installed in the cellar before the building is put up? The boiler is in the finished building, and in order to remove it later either the boiler has to be taken apart or a portion of the wall in the side of the building has to be torn down in order to remove the boiler from the house. I believe that the trypan blue enmeshed in the enamel is somewhat in the same condition, that is, it is so large it cannot be removed. In fact, the size of the

[7] Here Dr. Bödecker appears to have misunderstood the situation, as may be seen from the following quotation (Gies: *Journal of Dental Research*, 1926, vi, pp. 146–147): "The enamel protein is stained deeply by trypan blue. Some years ago the present writer [Gies] demonstrated, in dogs, that *while enamel is being produced, trypan blue, after its injection into the circulation, is absorbed into the enamel*, and is permanently retained; but that after enamel has been formed, trypan blue does not pass into it from the circulation, although the pigment enters the dentin. Since injected trypan blue is removed [directly or by chemical change] from all stained parts of the living body except dental enamel (after its passage into enamel under the special conditions just stated), it is probable that the degree of true *nutritional* transformation, as distinguished from simple mechanical change, in enamel *after its production*, is very slight if not wholly negative. If the enamel matrix is entirely inert chemically and devoid of nutritive influence, it is possible nevertheless that physical (mechanical) transmission of ions and molecules on or in its framework, distinct from physiological (selective) movement but inaugurated or controlled by conditions external to the matrix, might account for important effects on the enamel. Changes of this character appear to be the kind that Dr. Bödecker has projected but which he terms nutritional. Dr. Bödecker's very significant findings may lead to important discoveries, especially as to the origin and the prevention of dental caries. Such developments will be accelerated rather than retarded by searching scrutiny of all hypotheses regarding the function and dynamic relationships of the organic matrix."—[*Ed.*]

channels in the enamel and even those in the dentin are exceedingly minute, they are almost in fact at the border line of microscopic observation. Therefore, the amount of fluid as well as the rapidity of movement of fluid can only be minimal.[7]

As to Dr. Gies' suggestion for an experiment in this connection, I think it is valuable. My first plan, my first job was to find the channels in the enamel and dentin, and now we will go on and try to find whether there are any connections with the body lymph.

E. C. McBeath, D.D.S., M.D. (*New York City*): In carrying on our work on the nutritional phase of the problem of caries, to determine cause and means of prevention, we decided it would be well to adopt if possible some means to determine accurately the conditions as we find them from time to time, so that they could be uniformly recorded by different investigators. Of course, we record routinely any changes in the color and consistency of the carious dental tissue. However, we thought it advisable to devise, if possible, an instrument to measure the depth of a cavity. Drs. Bödecker and Leonard and I visualized our ideas. We submitted them to Mr. Crowther, in charge of the machine shop at the Dental School of Columbia University, and he gave us this instrument.

It consists of two parts: one for measuring the depth of the cavity, the other consists of a base and a receptacle for the first part. The part used for measuring the depth of a cavity consists of a brass cylinder, at the end of which and at right angles to it is a short brass tube, through the center of which passes a small steel needle. The long cylinder is hollow, and inside this cylinder are a weak spring and a metal rod. There is a slight contact between the end of the metal rod and the needle, creating just enough friction between the two to permit the needle to remain in the position to which it is forced after being introduced into the cavity. I have attempted to adopt a technic we could use at each examination, resting the instrument on the lingual and buccal cusps, each time in the same position as at the preceding examination. The second part consists of a metal base on which is mounted a vernier scale. Opposite the scale is an arm, which serves as a receptacle for the measuring instrument. Near this is an electric battery and lamp, connected with the arm; when connection is made between the measuring needle and the base of the arm, after adjusting the vernier scale, the lamp is lighted and the measurement is taken at that point. The scale measures 0.025 inch. It is the first instrument of its kind that I have seen.

Albert M. Judd, M.D. (*New York City*): It is indeed a great privilege to be present at such a representative gathering of a sister profession as I see here tonight. For this courtesy I am indebted to my friend, Dr. Waddell, and I thank him. I did not have the privilege, as did Dr. Gies, of reading Dr. Hawkins' paper so that I might prepare a discussion, but I could easily see how you enjoyed the discussion by Dr. Gies. Dr. Gies reads—Dr. Gies thinks—and when he puts his reading and his thoughts into words, he has made you all think, I am sure.

In my work at the Madison Hospital, I have become interested in the calcium and phosphorus contents of the blood—not of the saliva—from three standpoints. (1) From that of my obstetric work: there is a very old saying among the laymen that every child means the loss of a tooth. There may be more than a grain of truth in that. Of course, we all know that pregnancy brings greater requirements for calcium for both mother and expected child. (2) I have become particularly interested in the calcium and phosphorus contents in women who, coming to me, give a history of repeated miscarriages, where we have been able to rule out syphilis, but have had to fall back upon hyperthyroidism or some endocrine explanation. We find that there is a diminished content of both calcium and phosphorus; and that, by their administration in the form of medication and diet, we can in a great many cases carry a subsequent pregnancy to full fruition. (3) In my gynecological work I find it helps me in the major surgery of gynecology, such as prolonged bleeding from fibroids or fibroses uteri, to administer calcium previous to operation, thereby making the convalescence of that patient much better than otherwise it would be.

There is a condition that has bothered me for the last fifteen months, about which you as dentists may not be aware. Your papers, your essayists, may have helped me in a solution of this problem. During the past winter at least ten girls came to me complaining of a terrible inflammation in the vagina, with a discharge, some of them having been treated for a period of three or four months for gonorrhea—why, I don't know. Whether the men who previously saw these cases have mistaken a Diplococcus extracellular for a gonococcus, I do not know. But in these cases I found a so-called Trichomonas vaginalis with an extremely acrid vaginal secretion. It may be that in these cases, from what I have heard tonight, further investigation on my part may teach me how so to change the flora of the vagina that I may be able to overcome and relieve such cases, the treatment of which, at present, is very discouraging. I have been using various things. I have even scrubbed the vagina with green soap and tried a drying process. The treatment I am using at present is pyroligneous acid, then drying the

vagina and packing it with Lassar's paste. In a few of the cases, I have been able to remove the symptoms. The use of a lactic-acid douche has been very helpful. I used it in the proportion of two and one-half teaspoonfuls to two quarts of water.

There is a certain type of operative work, both abdominal and vaginal, where every time one uses a needle, the needle puncture will bleed. In these cases there is a deficient calcium content in the blood, but by proper prolonged treatment before operation, the operative technique can be made easier thereby. You probably all know (gall-bladder surgery was spoken of here tonight) that the administration of calcium previous to an operative procedure upon an old chronic gall bladder, particularly if jaundice is present, makes the operation much easier and prevents a great deal of oozing.

Dr. Hawkins (*in conclusion*): I want to thank Drs. Gies, Bödecker, and Judd for the comments on the paper. If Dr. Gies has found all the "bugs" that may be present in the paper, I will be very happy. For the last two and one-half years, I have read an average of two papers per month on this subject, and nearly all those commenting upon these papers have found additional "bugs." The result is that there has been quite a collection. If I could limit myself to the things Dr. Gies has suggested, I believe it would not take very long to run them down and make suitable explanations.

In regard to the people in the Hebrides, who seem to be on a diet that is more or less opposite to that which is recommended: I believe there are two things in common in the two diets. In that of the Hebrides there was a high-protein content. In the diet of the Orphans Home, we had a moderate, but I believe, adequate amount of meat protein, and eggs, which, I believe guarantees an adequate amount of phosphate. So I believe the two diets also had adequate phosphorus in common. In the Hebrides, we find liver entering very largely into the diet, which means a high-vitamin D content. There is a high concentration of that vitamin in the livers of animals. In the diet as carried out at the Los Angeles Orphans Home, while perhaps not made a part of this paper and perhaps not suitably emphasized, the vitamin-D content was considered, inasmuch as a very high-butter and high-egg content was always recommended, and liver once a week. So I believe we have, in the two diets, the two most important essentials in the control of dental decay; namely, an adequate amount of phosphorus, and an adequate amount of vitamin D. I believe that, in the case of those in the Hebrides, their saliva tests would show that they have acid mouths. If the calcium

content of the diet of these people had been high or fairly high, the salivary deposits would probably have been excessive, as the phosphorus and vitamin-D contents were excessive. On account of the high-phosphorus diet recommended in the Los Angeles Home, we had heavy salivary deposits —greater than when we started. I believe this was due, in part, to the fact that less care was used in tooth brushing. We know that an organic matrix seems to be a factor in the production of salivary deposits. The daily amounts of orange juice or cod-liver oil fed at the Los Angeles institution per child, for those who were put on that diet, was about half-a-glass of orange juice or about three teaspoonfuls of cod-liver oil.

I heartily agree with Dr. Gies that additional research would seem to be indicated. I believe, no matter how carefully any work is done, there is some opportunity for additional work in developing certain sidelights. So far as my own work is concerned, I fully appreciate that additional work would be of great value in establishing certain phases that are now obscure. I think that my paper was fairly clear in indicating that I personally believe the protection of the tooth comes from without. I do not feel that it is absolutely impossible that there might not be some protection from within, but I believe the major protection came from without through the saliva. That is the way I feel from my own results—my own experimentations. As far as the bathing of the tooth with saliva is concerned, the salivary bath is not uniform because teeth approximate each other, and deep pits and deep fissures also naturally make it impossible for saliva to give the same concentration or the same neutralizing value to a pit and a fissure that it would to a smooth surface. For that reason, I believe—we all know it from our observation—the weak points are where the teeth always or almost always fail to receive protection from the saliva, i.e., in the pits and fissures, or where the free flow of saliva is somewhat obstructed.

In regard to the changes in the salivary pH.: what I have suggested as being due to buffer action has been the hardest phase of my work to analyze. I have called in innumerable biochemists. I have put the problem before men of considerable standing, and nearly everyone deliberately evaded a direct answer. We have tried to eliminate the CO_2 factor by centrifuging, and by heating our samples. We found that the heated and unheated samples, or the centrifuged samples, if placed at the same time as those that are untreated, will reach approximately the same endpoint on standing. We have tested the possibilities of urea-breakdown as an explanation of the change, but that has been discarded as having probably no effect. We thought also of the fact that on standing some chemical changes might take place, so we took fresh samples of saliva, noted the change and the pH on

standing for one hour, then allowed the rest of that same sample to remain for two days at laboratory temperature and then determined its pH, and found that within very narrow limits the pH values of the samples were the same whether recorded immediately or after two days.

We have tried to explain it by other means, but we have come back to the same old problem; it seems that it must be the action of the indicator upon some chemical constituent in the saliva and combining with it. All of the indicators were either weak organic acids or bases. We know that acids or bases combine with acid salts or alkaline salts as the cases may be. We can see, therefore, why and how certain changes might take place.

While I do not know that I have completely answered all questions that Drs. Gies and Bödecker have raised, I feel that research has to be divided into two distinct parts—observation and interpretation, as Dr. Gies has mentioned. Our greatest discrepancy is in the interpretation of the results. Personally, I believe that I could take a group of 1000 children and carry them for three years, using the graph as a means of indicating their chemical status and delivering those children at the end of three years with an incidence of decay of not over 5 percent. I believe I could greatly improve on the 5 percent, and would not feel satisfied unless I could carry them through with at least that small incidence.

I would like to comment on Dr. Judd's question as to the bacteriological flora in the vagina. I have had no particular experience on vaginal secretions. I have had a great many cases of cystitis, however, and find that, in the cases I have tested, there was always an alkaline urine. When an acid urine is secreted in these cases, I find a marked improvement in the cystitis.

INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH

Scientific Proceedings of the New Haven Section
February 12, 1931

WILLIAM G. DOWNS, JR., M.A., D.D.S., Ph.D., *Editor*

Meeting held at the Laboratory of Oral Pathology, Yale Medical School, New Haven, Conn., February 12, 1931. *Chairman:* William G. Downs, Jr.

Scientific program

I. Incidence of dental caries among the Chinese. *Bert G. Anderson, D.D.S.* 235
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Abstracts

I. INCIDENCE OF DENTAL CARIES AMONG THE CHINESE

Bert G. Anderson, D.D.S., Department of Surgery, Yale Medical School, New Haven, Conn.

Showed the incidence of dental caries by teeth and tooth surfaces in 975 Chinese individuals. Judged by reports of other workers, caries is much less general among the inland Chinese than among Americans. Full report and tables are to be published later.

II. TWO UNUSUAL ORTHODONTIC CASES

A. W. Crosby, D.D.S., Department of Surgery, Yale Medical School, New Haven, Conn.

The first case: a marked open bite in the region of the temporary molars. Later the permanent bicuspids did not respond to the usual treatments. Functional treatment with Ferris' Odonto-Bell plus

muscle massage gave excellent results, which are apparently permanent. The second case illustrated the possibilities in badly jumbled dentitions with marked rotation of the teeth. Marked improvement in the general physical condition of both patients is shown by the photographs.

III. A CASE OF MANDIBULAR HYPEROSTOSIS

J. P. Pigott, D.D.S., Department of Surgery, Yale Medical School, New Haven, Conn.

A case of marked unilateral lingual enlargement in the mandibular bicuspid region, similar probably to the torus palatinus on the palate. This condition, described by Hrdlicka in the Eskimo and Indian, is said to be very rare among whites.

IV. CORRELATION OF AUTOPSY DATA

S. S. Arnim, B.S., D.D.S., Department of Pathology, Yale Medical School, New Haven, Conn.

Dental entities and anatomical autopsy diagnoses were the basis for an attempted correlation of the data from 65 autopsies performed in this department. The need for more complete clinical histories and better understanding of the requirements of the statistical method were stressed.

V. EFFECTS OF VARIOUS DIETS ON RATE OF GROWTH OF RAT TEETH

William G. Downs, Jr., M.A., D.D.S., Ph.D., Department of Pathology, Yale Medical School, New Haven, Conn.

A preliminary report, suggesting that the rate of growth is accelerated by high-protein and high-carbohydrate diets, affected only slightly by high-fat diets and inhibited by low-total-salt, low-PO_4 and low-Ca diets.

THE WHITE HOUSE CONFERENCE ON CHILD HEALTH AND PROTECTION[1]

A Report

MILO HELLMAN, D.D.S.

Member of Section I-A on Growth and Development

When President Hoover's intention of doing something for the child population of the United States was given its first publicity, those of the general public who paid any attention at all received it with mixed feelings of indifference, doubt and apprehension. Whether this attitude has changed, I don't know. But from personal experience I am quite certain that most people hardly know anything about it and, of those who do know, some are not interested in it at all and others suspect some ulterior motives. For this reason I accepted the invitation to report the matter, at least before the dental profession. In doing this I feel that some justice will be done to an undertaking which is as stupendous in magnitude as it is unequivocal in significance. From the point of importance a report of this sort should be given more time than is available this evening. But neither the program committee nor I could, under the circumstances, do more than is now arranged for. Brevity will therefore have the precedence over thoroughness.

The White House Conference on Child Health and Protection had its origin in 1929. President Hoover in his opening address told us:

"Something more than a year ago I called together a small group of representative men and women to take the initial steps in organization of this conference on child health and protection. Under the able chairmanship of Secretary Wilbur, and the executive direction of Dr. Barnard, organization was perfected and enlarged until by the Fall of last year something

[1] Read at a meeting of the New York Academy of Dentistry, Yale Club, New York City, January 22, 1931.

over 1,200 of our fellow citizens were enlisted from every field of those who have given a lifetime of devotion to public measures for the care of childhood. These skillful and devoted friends of children have given unsparingly and unselfishly of their knowledge and experience in the problems involved. Their task has been magnificently performed, and today they will place before you such wealth of material as was never before brought together.

"The fundamental purpose of this conference is to set forth an understanding of those safeguards which will assure to them [the children] health in mind and body. There are safeguards and services to childhood which can be provided by the community, the state, or the nation—all of which are beyond the reach of the individual parent. We approach these problems in no spirit of diminishing the responsibilities and values or invading the sanctities of those primary safeguards to child life—their homes and their mothers. After we have determined every scientific fact, after we have erected every public safeguard, after we have constructed every edifice for public education or training or hospitalization or play, yet all these things are but a tithe of the physical, moral and spiritual gifts which motherhood gives and home confers. None of these things carry that affection, that devotion of soul, which is the great endowment from mothers. Our purpose here today is to consider and give our mite of help to strengthen her hand that her boy and girl may have a fair chance.

"Our country has a vast majority of competent mothers. I am not so sure of the majority of competent fathers. But what we are concerned with here are things that are beyond her power. That is what Susie and John take on when out from her watchful eye. She cannot count the bacteria in the milk; she cannot detect the typhoid which comes through the faucet, or the mumps that pass around the playground. She cannot individually control the instruction of our schools or the setting up of community-wide remedy for the deficient and handicapped child. But she can insist upon officials who hold up standards of protection and service to her children—and one of your jobs is to define these standards and tell her what they are. She can be trusted to put public officials to the acid test of the infant mortality and service to children in town—when you set some standard for her to go by.

"These questions of child health and protection are a complicated problem requiring much learning and much action. And we need have great concern over this matter. Let no one believe that these are questions which should not stir a nation; that they are below the dignity of statesmen and governments. If we could have but one generation of properly born, trained, educated and healthy children, a thousand other problems of

government would vanish. We would assure ourselves of healthier minds in more vigorous bodies, to direct the energies of our nation to yet greater heights of achievement. Moreover, one good community nurse will save a dozen future policemen.

"Our problem falls into three groups: First, the protection and stimulation of the normal child; second, aid to the physically defective and handicapped child; third, the problems of the delinquent child."

The magnitude of the problem will be realized when it is learned that, according to one of the committee reports, out of 45,000,000 children—

35,000,000 are reasonably normal
6,000,000 are improperly nourished
1,000,000 have defective speech
1,000,000 have weak or damaged hearts
675,000 present behavior problems
450,000 are mentally retarded
382,000 are tubercular
342,000 have impaired hearing
18,000 are totally deaf
300,000 are crippled
50,000 are partially blind
14,000 are wholly blind
200,000 are delinquent
500,000 are dependent

The manner in which this problem is to be coped with will be seen in the plan of organization of the White House Conference. The various branches of knowledge on which more information is sought are divided into four primary sections:

Section I. Medical Care
Subsection A has its special problem: Growth and Development
Subsection B is concerned with the problem of Prenatal and Maternal Care
Subsection C is concerned with Medical Care for Children
Section II. Public Health Service and Administration
Subsection A. Public Health Organization
Subsection B. Communicable Disease Control
Subsection C. Milk Production Control

Section III. Education and Training
- Subsection A. The Family and Parent Education
- Subsection B. The Infant and Preschool Child
- Subsection C. The School Child
- Subsection D. Vocational Guidance and Child Labor
- Subsection E. Recreation and Physical Education
- Subsection F. Special Classes
- Subsection G. Youth outside Home and School

Section IV. The Handicapped
- Subsection A. State and Local Organization for the Handicapped
- Subsection B. Physically and Mentally Handicapped
- Subsection C-1. Socially Handicapped—Dependency and Neglect
- Subsection C-2. Socially Handicapped—Delinquency

These subsections are further divided into committees and subcommittees. Thus, dentistry is represented by a subcommittee under Section I, Subsection A.

The viewpoints represented in the deliberations and the angles of approach to the problems at the sectional meetings are diverse and manifold. There are in Section I—Medical Care, for example, representative authorities and recognized specialists in anatomy, physiology, normal variations in physical development, immunology, roentgenology, normal nutritional requirements, normal variations in mental development, heredity, statistics and factors influencing growth and development. It is therefore quite obvious that, if the outcome will depend upon the proper selection of talent, success is assured. Moreover, as stated by the Chairman of Section I: "We have here a unique opportunity for coöperation and for learning the other fellow's viewpoint and problem. Heretofore there have been many groups—scientists, physicians, psychiatrists and psychologists, nurses, social workers, dentists, educators, juvenile court workers, and many others—all laboring in their own fields, all with the same underlying aims, but ignorant of the details of the work of the others and its relationship to theirs. This has all too often led to a lack of sympathy which has, at times, obstructed achievement.

"One of the most encouraging results of the conference would be a

greater give and take among the workers, a spirit that says 'Time can be saved and the child better served, if we give one another the benefit of our specialized knowledge just where the other's training falls short.' Such unity in itself would be a portent of greater achievement." How different is the spirit of these thoughts from what is going on right among us now! If I may just mention one thing, there is a good bit of dust raised by the Oral Hygiene Committee of Greater New York, because the result of an investigation on dental caries by the American Child Health Association revealed certain facts which are contrary to previous findings. Instead of looking for the reason for such differences in results, fault is found with the investigators.[2] I just mention this in contrast to the spirit prevailing among the representative authorities at the White House Conference.

The care with which the various tasks were approached will be seen in the following statement made by the Chairman of Section I-A. "Knowledge of growth and development of children is extensive in some respects but meagre in others. It is important to recognize the gaps which exist and admit ignorance, rather than to theorize with unwarranted assurance." The greatest part of the task was first to obtain an appraisal of what is known about child health and protection, of what is not known and of what should be known. To this end everyone connected with this undertaking had to submit an authoritative report supported by reliable proof derived from scientific investigations in his or her special field of work. These reports will be published in toto, but from the scientific facts thus gathered special recommendations will be made which are to serve as guiding principles for the future.

Of course, the most important, interesting and significant part of the task was the matter concerned with these reports. The dental subcommittee, for instance, had to do its share of the work. But a year had passed and nothing was forthcoming. Questionnaires were sent out to the educational leaders in dentistry with the object of gathering sufficient information for the preparation of such a report. I suppose many of those present also received copies of these questionnaires. But curiously enough no information could be gathered that

[2] Another paper on this subject, by the Oral Hygiene Committee, is published in this issue: *Journal of Dental Research*, 1931, xi, p. 305.—[*Ed.*]

way. The committee was in despair. Advice from certain quarters outside the dental profession was of help. The dental committee at one of the various meetings was informed that the material needed for their report was available and could be obtained by return mail, if a certain individual in the dental profession was consulted. This was soon accomplished. A very lovely letter was sent to him by the chairman of the dental committee requesting all the data available on the subjects of growth and development from the dental aspect. Of course, the importance of the service thus to be rendered to the conference in the name of dentistry was sufficiently emphasized. The request was granted and all published data were submitted to the committee. The unpublished material, however, though needed was withheld. There was then no way out but to enlist the coöperation of its holder. So in July an offer of appointment to the White House Conference came. I accepted. But no sooner did I accept than I was commissioned to submit the necessary report and that in a month's time. I could just then see my long-looked-for vacation go up in a huge cloud of smoke. The thing, however, had to be done and it was done. My report dealt with the "Development of the Face, Jaws and Dentition." But I felt that an account also had to be given of the "Eruption of the Teeth" as a measure of development. So the report really turned out to be twins. Of the other committee men, one was to give a report on "The Present Status of Dental Caries" and the other on "Diseases and Abnormalities of the Mouth." At the Conference in November mine were the only reports submitted. But, curiously enough, in the program of the dental subcommittee meetings the twins were separated. One was credited to me and the other to someone else. Of course, it was both a surprise and an interesting experience. It showed, nevertheless, how certain jobs are held on to under certain adverse circumstances.

It is very interesting that, even in events of such significant nature, small personal things do crop out. For instance, after the President's address everyone present seemed to be deeply impressed; at least I was. But on leaving the hall, I met an old friend who happens to be a member of the United States Congress. He is a Democrat. I don't know what he thinks I am. When he saw me, he greeted me as follows: "Hello, Hellman, how did you like the President's speech?

Didn't he speak just like a Republican?" At first I was confused, but fortunately, I do possess a sense of humor and smiled. Contrast this with the following expression of one of the delegates: "I have learned, from those who are close to him and know his heart, that one great theme of his life is in this great conference that he has called together. I felt last night, as I am sure you did, that not one single word uttered was simply an expression of belief, but the throbbing of a great human heart of these 120,000,000 of people over whom he presides, and for that other million of millions reaching out through all the lands of the earth to whom he has so wonderfully administered in the last twelve or fifteen years."

Whatever the personal feelings may be, I think that there is sufficient reason to be proud of a country and its administration for the initiation of a movement that may be of more far-reaching significance than we can at present even dream. The final recommendations have as yet not been made, because not all the reports had been submitted at the last meeting. All the material is complete, and the work of the chairmen is now going on feverishly. The literature assembled in the volumes to be published will be unique. It will furnish a source of knowledge for students the equal of which has not been known. The recommendations promise to be to the point, short, concrete, clear, of practical value and of direct benefit to the future citizens of our great country. A sample of what may be expected as the final outcome of the White House Conference is well illustrated by one of the conclusions reached by subsection C—Medical Care for Children—with direct reference to dentistry: "The practice of oral hygiene and reparative dentistry, carried on for a number of years, while improving general health, has failed to decrease the incidence of dental caries among children. Such work should be continued and extended, but intensive experimental and clinical work on a large scale should be undertaken to definitely determine the part played by mineral metabolism as influenced by dietary procedures in the incidence of dental caries." Similarly, a subcommittee of Section II dealing with Public Health Service and Administration "calls attention to the fact that there are certain phases of public health work which are especially well adapted for developing a constructive program of coöperation between health agencies and the physicians and dentists of a com-

munity. The state and local health departments should advise with the medical and dental professions in determining such programs. The state medical and dental associations should appoint, for the benefit of the state health officer, advisory committees approved by their legislative bodies. Likewise, the district or county organizations should appoint advisory committees for local health officers. These committees will prove invaluable in adjusting differences, stimulating interest and in securing coöperation on the part of practitioners. Practising physicians and dentists are, and should be, very important factors in safeguarding the health of the public."

In conclusion I should like to read to you "The Children's Charter," which has been so beautifully worked out and is to be included in the introduction to the reports.

"President Hoover's White House Conference on Child Health and Protection recognizing the rights of the child as the first rights of citizenship pledges itself to these aims for the children of America.

"I. For every child spiritual and moral training to help him to stand firm under pressure of life.

"II. For every child understanding and the guarding of his personality as his most precious right.

"III. For every child a home and that love and security which a home provides; and for that child who must receive foster care, the nearest substitute for his own home.

"IV. For every child full preparation for his birth, his mother receiving prenatal, natal, and postnatal care; and the establishment of such protective measures as will make child-bearing safer.

"V. For every child health protection from birth through adolescence, including: periodical health examinations and, where needed, care of specialists and hospital treatment; regular dental examination and care of the teeth; protective and preventive measures against communicable diseases; the insuring of pure food, pure milk and pure water.

"VI. For every child from birth through adolescence, promotion of health, including health instruction and a health program, wholesome physical and mental recreation, with teachers and leaders adequately trained.

"VII. For every child a dwelling place safe, sanitary, and wholesome, with reasonable provisions for privacy, free from conditions which tend to thwart his development; and a home environment harmonious and enriching.

"VIII. For every child a school which is safe from hazards, sanitary,

properly equipped, lighted, and ventilated. For younger children nursery schools and kindergartens to supplement home care.

"IX. For every child a community which recognizes and plans for his needs, protects him against physical dangers, moral hazards, and disease; provides him with safe and wholesome places for play and recreation; and makes provision for his cultural and social needs.

"X. For every child an education which, through the discovery and development of his individual abilities, prepares him for life; and through training and vocational guidance prepares him for a living which will yield him the maximum of satisfaction.

"XI. For every child such teaching and training as will prepare him for successful parenthood, homemaking and the rights of citizenship; and, for parents, supplementary training to fit them to deal wisely with the problems of parenthood.

"XII. For every child education for safety and protection against accidents to which modern conditions subject him—those to which he is directly exposed and those which, through loss or maiming of his parents, affect him indirectly.

"XIII. For every child who is blind, deaf, crippled, or otherwise physically handicapped, such measures as will early discover and diagnose his handicap, provide care and treatment, and so train him that he may become an asset to society rather than a liability. Expenses of these services should be born publicly where they cannot be privately met.

"XIV. For every child who is in conflict with society the right to be dealt with intelligently as society's charge, not society's outcast; with the home, the school, the church, the court and the institution when needed, shaped to return him whenever possible to the normal stream of life.

"XV. For every child the right to grow up in a family with an adequate standard of living and the security of a stable income as the surest safeguard against social handicaps.

"XVI. For every child protection against labor that stunts growth, either physical or mental, that limits education, that deprives children of the right of comradeship, of play, and of joy.

"XVII. For every rural child as satisfactory schooling and health services as for the city child, and an extension to rural families of social, recreational, and cultural facilities.

"XVIII. To supplement the home and the school in the training of youth, and to return to them those interests of which modern life tends to cheat children, every stimulation and encouragement should be given to the extension and development of the voluntary youth organizations.

"XIX. To make everywhere available these minimum protections of the health and welfare of children, there should be a district, county, or community organization for health, education, and welfare, with full-time officials, coördinating with a state-wide program which will be responsive to a nation-wide service of general information, statistics, and scientific research. This should include:

"(a) Trained, full-time public health officials, with public health nurses, sanitary inspection, and laboratory workers;

"(b) Available hospital beds;

"(c) Full-time public welfare service for the relief, aid, and guidance of children in special need due to poverty, misfortune, or behavior difficulties, and for the protection of children from abuse, neglect, exploitation, or moral hazard.

"For *every* child these rights, regardless of race, or color, or situation, wherever he may live under the protection of the American flag."

57 West 57th Street,
New York City

THE PROCESS OF REPAIR FOLLOWING TOOTH FRACTURE[1]

RUDOLF KRONFELD, M.D.

Research Department, Chicago College of Dental Surgery, Dental Department of Loyola University, Chicago, Ill.

Recently there have been reports on the process of repair in fractured teeth, by Gottlieb (6), Euler and Meyer (4), Siegmund and Weber (9), Greve (7), Szabo (10), Boulger (1), Kronfeld (8), Euler (3) and Figg (5). From these investigations we know that, after a traumatic injury to the root of a tooth, cementum is deposited on the fractured surfaces, and, under favorable conditions (absence of infection), the fracture may heal. All these publications, however, dealt with cases that were studied some time after the fracture had occurred, and there had already been a considerable formation of cementum. It seems desirable, therefore, to describe this particular case in which the fracture occurred shortly before the specimen was obtained, thus making it possible to study the first steps in the process of healing.

In studying serial sections through the teeth of a man of 38, who died of tuberculosis, we found that the cementum had been torn away from the dentin on the lingual side of the root of a lower central incisor. This type of tooth fracture, first described by Euler (3), originates in the following way: When a tooth is hit by a sudden blow that causes damage to the root, circumscribed areas of cementum, the connection of which with the fibre bundles of the periodontal membrane is stronger than the junction with the dentin, are torn from the root surface. Such splinters of cementum become displaced into the periodontal membrane—Euler (3, 4), Figg (5). This type of fracture of the cementum undoubtedly occurred in the case described above.

[1] Read at the ninth general meeting of the International Association for Dental Research, Hotel Peabody, Memphis, Tenn., March 21–22, 1931.

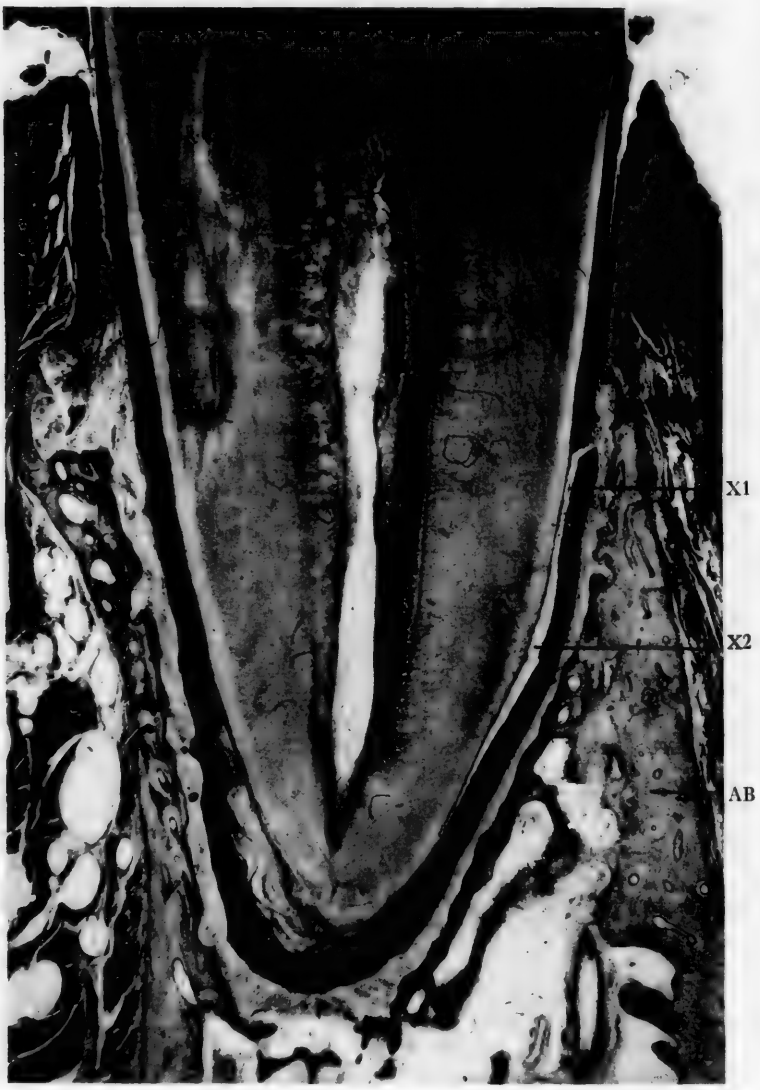

FIG. 1. LABIO-LINGUAL SECTION THROUGH LOWER CENTRAL INCISOR ROOT (×15)

AB—Alveolar bone. *X1*, *X2*—Crack between cementum and dentin on lingual side of root.

Shortly before the death of the patient, the incisor must have been subjected to a sudden thrust toward the labial side, thus causing a

FIG. 2. AREA X1 OF FIG. 1; UPPER END OF CRACK (×150)

D—Dentin. *C*—Cementum. *O*—Opening of crack, in connection with periodontal membrane, *PM*. *CT*—Connective tissue in crack. *R*—Resorption of alveolar bone.

tear in the cementum on the lingual side of the root (*fig. 1*). A general view shows that this tear extends over more than one half of the clinical root. The periodontal space appears smaller in this area than

on the opposite side of the root, probably because of the dislocation of the splinter. From investigations on old healed fractures of this type, we know that in such cases the periodontal membrane reassumes its former width over the fractured area—Boulger (1), Euler (3), Figg (5). In this particular case, this had not yet taken place.

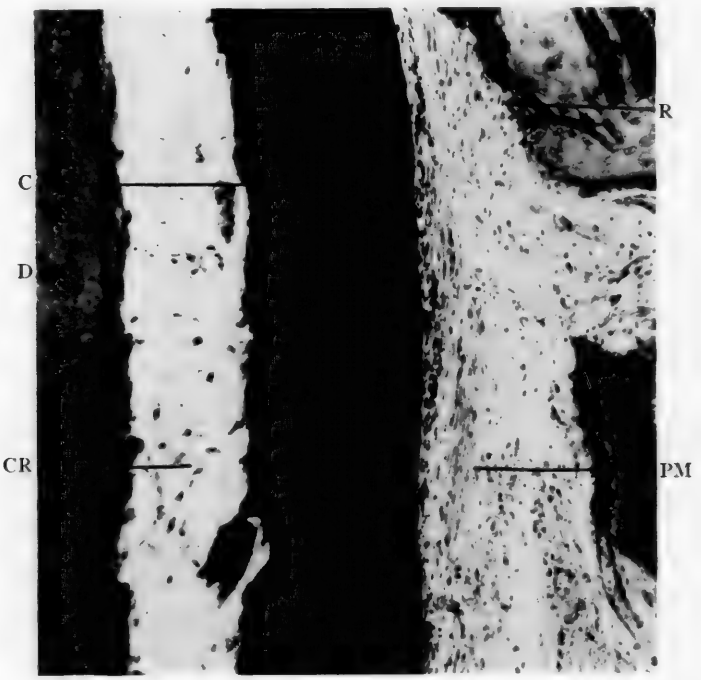

FIG. 3. AREA X2 OF FIG. 1: APICAL PART OF THE CRACK (×150)

D—Dentin. *C*—Cementum. *CR*—Crack containing coagulated lymph and loose network of large cells. *PM*—Periodontal membrane. *R*—Resorption of alveolar bone

However, there were indications of resorption on the inside of the alveolar bone, evidently tending toward a widening of the periodontal space and toward a restoration of a normal functional periodontal membrane in the area of the fracture.

Figs. 2–8 are higher magnifications of areas X1 and X2 in *fig. 1*. In *fig. 2* we see the upper end of the detached splinter of cementum.

The separation between dentin and cementum is complete, very small particles of cementum remaining connected with the dentin surface in only a few places. The crack is filled with connective tissue that proliferated from the periodontal membrane through the opening at

FIG. 4. HIGHER MAGNIFICATION OF FIG. 3 (×600)
D—Dentin. *C*—Cementum. *Fb*—Fibroblasts. *Fl*—Fibrillae of connective tissue

the upper end. This tissue shows the same structure as the periodontal membrane; it extends, however, only a short way into the crack. Toward the apex the density of the tissue and the number of cells are rapidly decreasing. In the middle part of the crack (*fig. 3*, area X2 of *fig. 1*), the space between dentin and cementum is filled

with a coagulated fluid (lymph) that appears light pink in the original specimen. In this fluid cells, apparently freely suspended, are connected with each other by fine protoplasmic processes. A higher magnification (*fig. 4*) shows that these cells are typical fibroblasts. They have large, round, or ovoid nuclei and several protoplasmic processes. The product of these cells, very fine connective-tissue fibrillae, are visible everywhere in the crack. In the direction of the apex the cellular elements in the fissure steadily decrease in number until

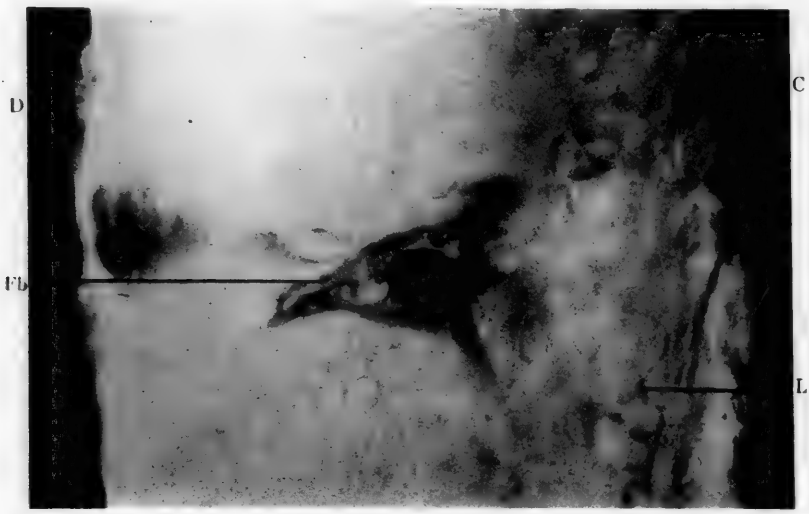

FIG. 5. HIGH-POWER PHOTOGRAPH OF APICAL PART OF CRACK (×1200)

D—Dentin. *C*—Cementum. *L*—Coagulated lymph. *Fb*—Fibroblast with protoplasmic processes.

finally, at the bottom of the crack, there is left only coagulated lymph without structure. This gives us a clear understanding of the process that took place between cementum and dentin in this small fissure. Immediately after the fracture, the crack became filled with lymph. Then the cells of the periodontal connective tissue, adjacent to the upper end of the crack, started to proliferate and grew into the lymph-filled space. Thus, we find in this space different stages of development of connective tissue: ripe tissue at the entrance of the crack; younger

tissue nearer the apex; still further down, single fibroblasts, as the cell proliferation into the lymph advanced. In *figs. 5–8* we see, under higher magnification, the condition of the cells in the lower part of the crack.

Fig. 5 was taken from the apical end of the crack. The space between dentin and cementum is filled with lymph, which shows a vesicular structure due to coagulation and fixation. In the middle of the

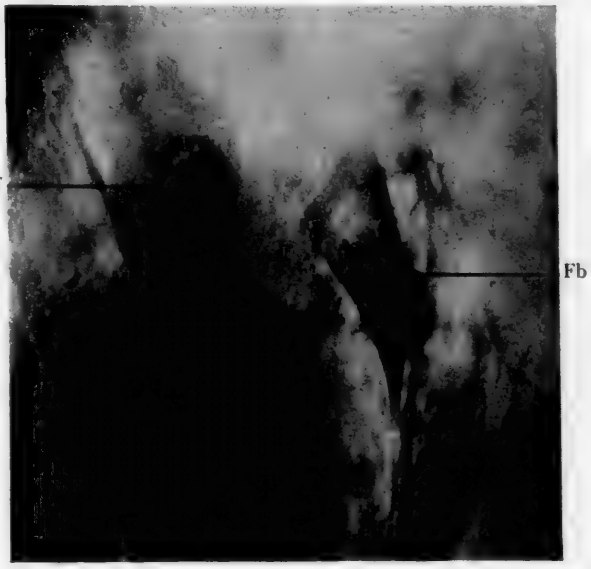

FIG. 6. WANDERING CELL AND FIBROBLAST IN APICAL PART OF CRACK (×1200)

W—Wandering cell with kidney-shaped nucleus and granulated protoplasm. *Fb*—Fibroblast.

fluid there is a single cell, a fibroblast, with protoplasmic processes in three directions. In fact, this cell has still more such processes, but it is not possible to reproduce in a photograph the three dimensions of such a cell. No connective-tissue fibrillae are as yet present in this particular area of the crack. A little farther up we find a few cells with the characteristics of wandering cells (*fig. 6*). They have kidney-shaped nuclei and regular round bodies of protoplasm. These cells, like all other cells in this area, appear in their "classic form."

They are free and undisturbed, developing in the lymph between two hard walls, and are not exposed to any mechanical influences, such as are normally present in the connective tissue (tension, lack of space). Near this wandering cell there is another typical fibroblast.

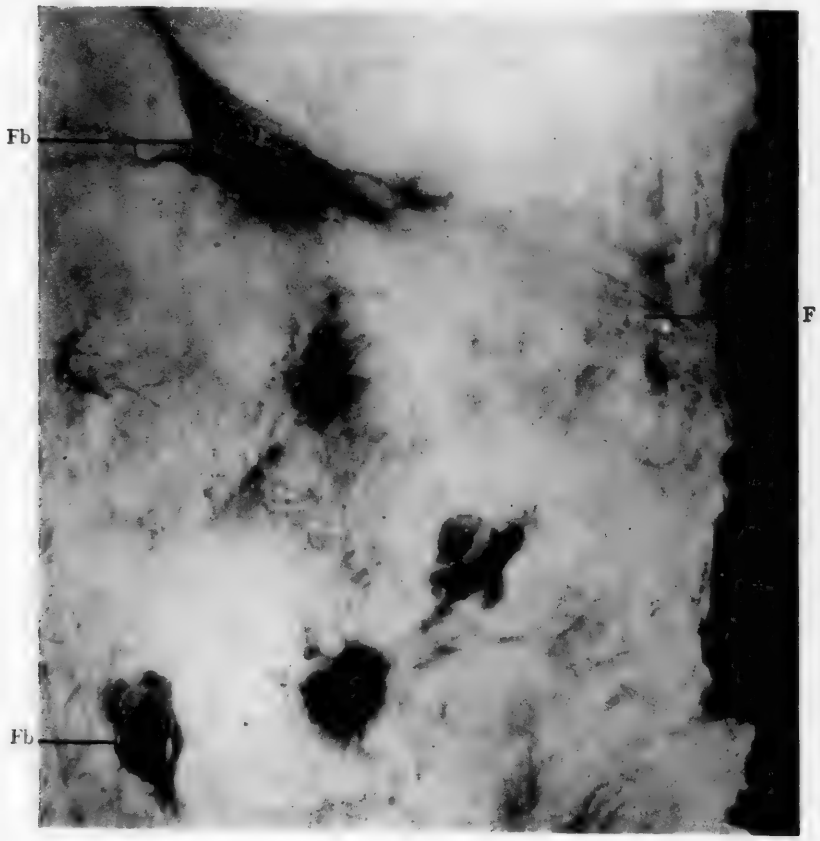

FIG. 7. FIBROBLASTS, *Fb*, AND FIBRILLAE, *F*, IN MIDDLE PART OF CRACK (×1200)

Still farther up, toward the entrance of the crack, the arrangement of the fibroblasts becomes denser (*fig. 7*). Between the cell bodies a fine fibrillar matrix represents the first step toward the formation of fibrous connective-tissue. The fibroblasts lie in different directions

(*fig. 8*). Some of the cell bodies are parallel to the course of the crack, and others are perpendicular to it. Accordingly, the bundles of

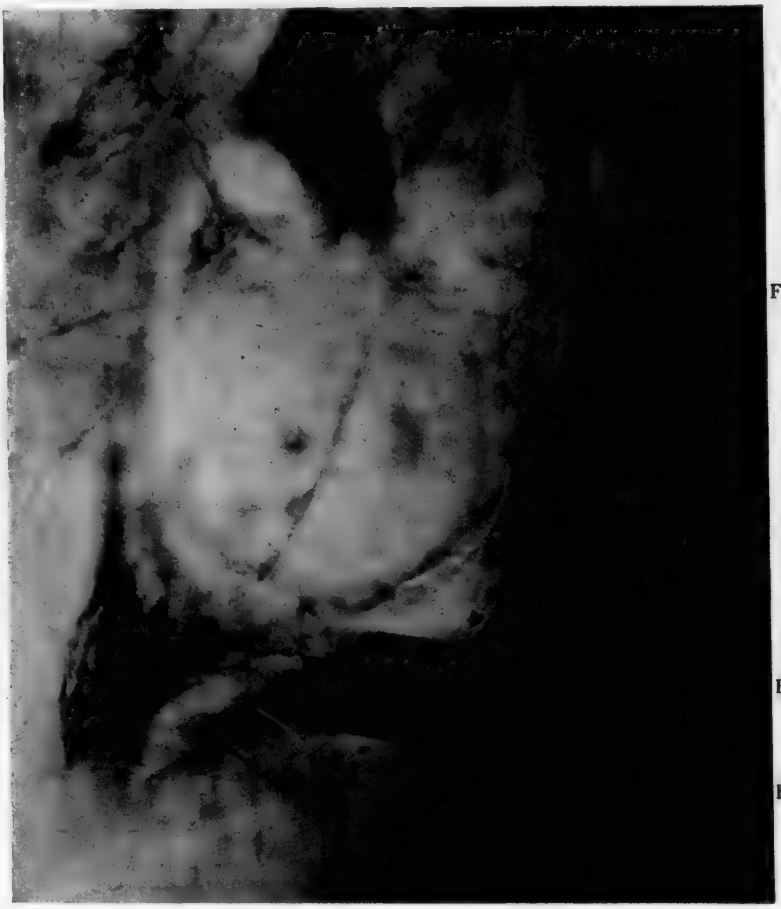

FIG. 8. FIBROBLASTS, *Fb*, AND FIBRILLAE, *F*, IN THE MIDDLE PART OF THE CRACK; FIBROBLASTS LIE IN DIFFERENT DIRECTIONS (×1200)

Fb1—Fibroblast lying parallel to direction of crack. *Fb2*—Fibroblasts lying at right angles to crack. *F*—Fibrillae.

fibrillae are also arranged in different directions, thus forming a dense network across the fissure. The individual fibroblasts in this area

are considerably larger than the corresponding cells in the other connective tissue, as, for instance, in the periodontal membrane. This is in accordance with observations in experimental tissue-cultures, in which the single cells are larger than the same type of cell in the original body-tissue.

In the entire series of slides, it was not possible to find any blood vessels in the crack. It seems that a network of fibrillae and fibroblasts is formed first, and capillaries then sprout into it.

Summary. After a traumatic fracture of a part of the cementum, the resulting crack was filled with lymph. From the periodontal connective-tissue, fibroblasts and wandering cells grew into this lymph in the manner in which such cells proliferate in the culture medium of a tissue culture. The fibroblasts formed a network of fibrillae. In this way connective tissue and cementum were built, and the fracture was repaired.

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HISTOLOGIC STUDY OF A HYPERTROPHIED PULP[1]

EARL P. BOULGER, D.D.S., L.D.S.

Chicago, Ill.

The exposure of a vital pulp to the irritating influences in the mouth usually causes the loss of the pulp with the attending sequellae. Instances do occur, however, when an exposed pulp is highly resistant to such influences and adjusts itself to its new environment. Then, a chronic pulpitis of long standing is likely to develop, and eventually the exposed surface of the pulp is found to be epithelized. Several such cases of polypus of the pulp, with an epithelial covering, have already been reported (2, 3). However, there is no description of the relationship between the epithelium and the hard tissues of the tooth, and it is from this particular viewpoint that we consider our case to be of interest. The specimen under consideration was a broken-down, lower, second bicuspid in a patient about twenty years of age. The pulp was a proliferation that filled the pulp chamber, and extended beyond the pulpal wall into the cavity. The pulp tissue, however, rested entirely upon the dentin, and did not reach the surrounding soft structures. The tissue was slightly responsive to stimulus, and presented an appearance simulating gum tissue. The tooth was extracted, decalcified, imbedded in celloidin, and sectioned in serial sections.

Fig. 1 shows a general view of the pulp canal and the hypertrophied pulp tissue. The pulp canal is narrowed by the deposition of secondary dentin (*SD*), which is laid down in irregular masses and is in various stages of calcification. The pulp tissue that extends into the cavity is covered by a layer of stratified squamous epithelium (*E*), and the inflammatory condition of the pulp is indicted by the presence of round-cell infiltration.

[1] From the Research Department (Director: Rudolf Kronfeld) of the Chicago College of Dental Surgery, Dental Department of Loyola University. Read at a meeting of the Chicago Section of the International Association for Dental Research, April 30, 1931.

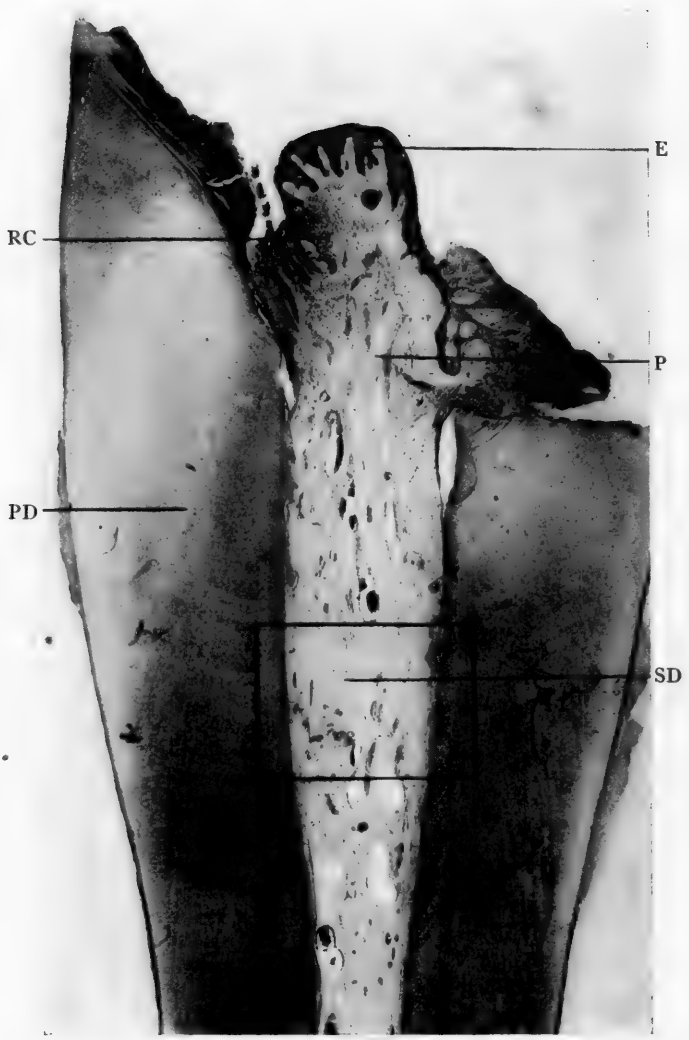

FIG. 1. BROKEN-DOWN, LOWER, SECOND BICUSPID

E—Epithelium covering hypertrophied pulp tissue. *P*—Pulp tissue. *SD*—Secondary dentin. *PD*—Primary dentin. *RC*—Round-cell infiltration in pulp.

A higher magnification of area *SD* (*fig. 2*) shows more clearly the deposition of secondary dentin along the wall of the canal. Embedded in this dentin are calcified nodules of pulp tissue (*N*), which evidently underwent degeneration, became calcified, and were enclosed in the

FIG. 2. AREA *SD* IN FIG. 1

PD—Primary dentin. *SD*—Secondary dentin deposited in canal. *P*—Pulp tissue. *N*—Nodules of calcified pulp-tissue embedded in secondary dentin. *O*—Remnants of odontoblastic layer. *C*—Pulp canal reduced in width by secondary-dentin formation.

secondary dentin produced by the pulp. The large masses of secondary dentin have greatly reduced the size of the canal (*C*), thus indicating nature's effort to seal the pulp from outside irritants. Remnants of the odontoblastic layer can be seen at *O*.

Fig. 3 shows a high magnification of the secondary dentin and adjacent pulp tissue. The secondary dentin (*SD*) is characterized by the presence of only a few irregular tubules, whereas the primary dentin (*PD*) has a regular tubular structure. A definite sharp border exists between these two types of dentin.

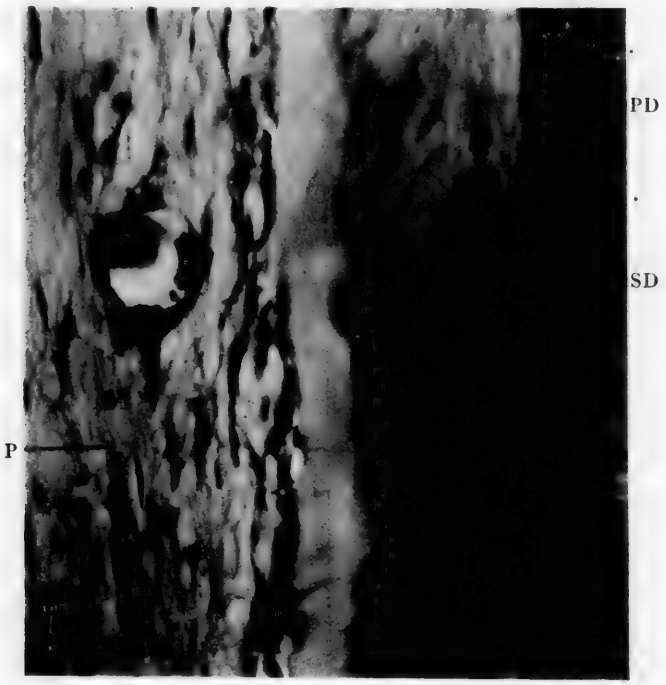

FIG. 3. HIGHER MAGNIFICATION OF *SD* IN FIG. 2
PD—Primary dentin. *SD*—Secondary dentin. *P*—Pulp tissue

As already mentioned, epithelium, which in its structure is identical with that of the gum tissue, covers the exposed and proliferated pulp (*fig. 4*). At the point where this epithelial covering comes in contact with the dentin, it forms an epithelial attachment that extends down into the root canal (*EA*), and is attached to the wall of dentin. On one side of the root canal, a typical pocket has been formed between the epithelium and dentin (*P*). At *H*, where the epithelium is well

hornified, this layer is due to the friction on this area in mastication (Orban, 5).

FIG. 4. EPITHELIUM COVERING EXPOSED AND PROLIFERATED PULP

EA—Epithelial attachment to dentinal wall of pulp canal. *PD*—Primary dentin. *P*—Pocket between epithelium and wall of pulp chamber. *H*—Hornified layer of epithelium.

Further down, toward the bottom of the pocket (*fig. 5, P*), the epithelium is inflamed and in some instances has been completely de-

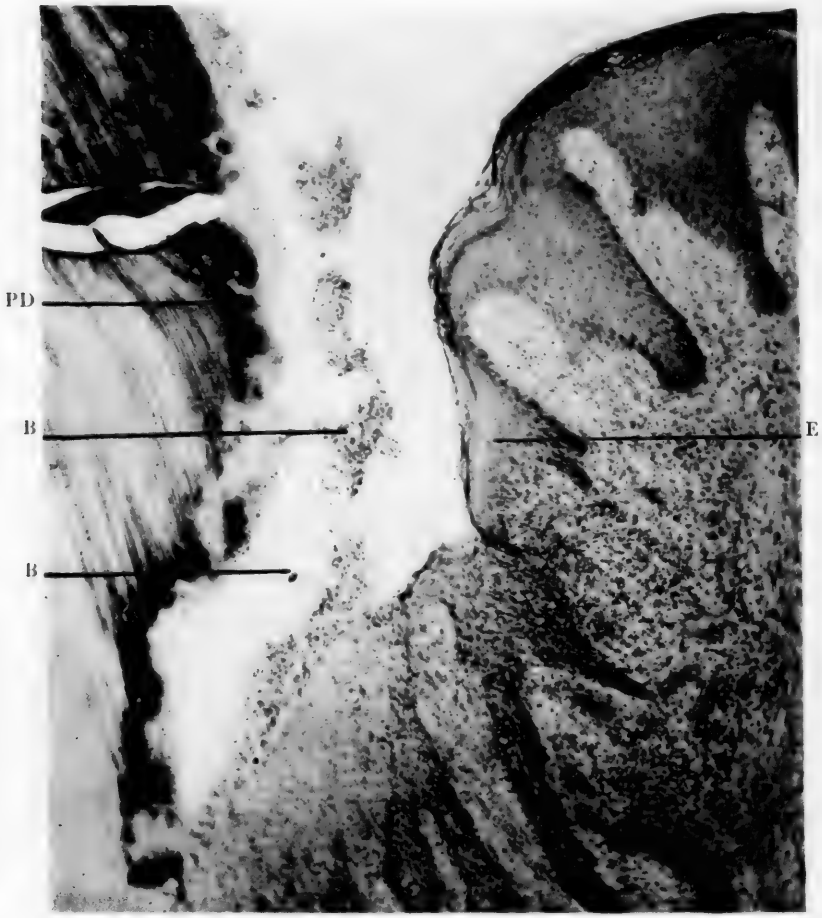

FIG. 5. HIGHER MAGNIFICATION OF POCKET *P* IN FIG. 4

E—Epithelium forming one wall of pocket *P*. *PD*—Decayed primary dentin that forms other wall of pocket. *B*—Debris, leukocytes, and bacteria lodged in pocket.

stroyed, thus permitting the inflammatory round-cells of the retro-epithelial pulp-tissue to appear in the pocket in the form of pus. The

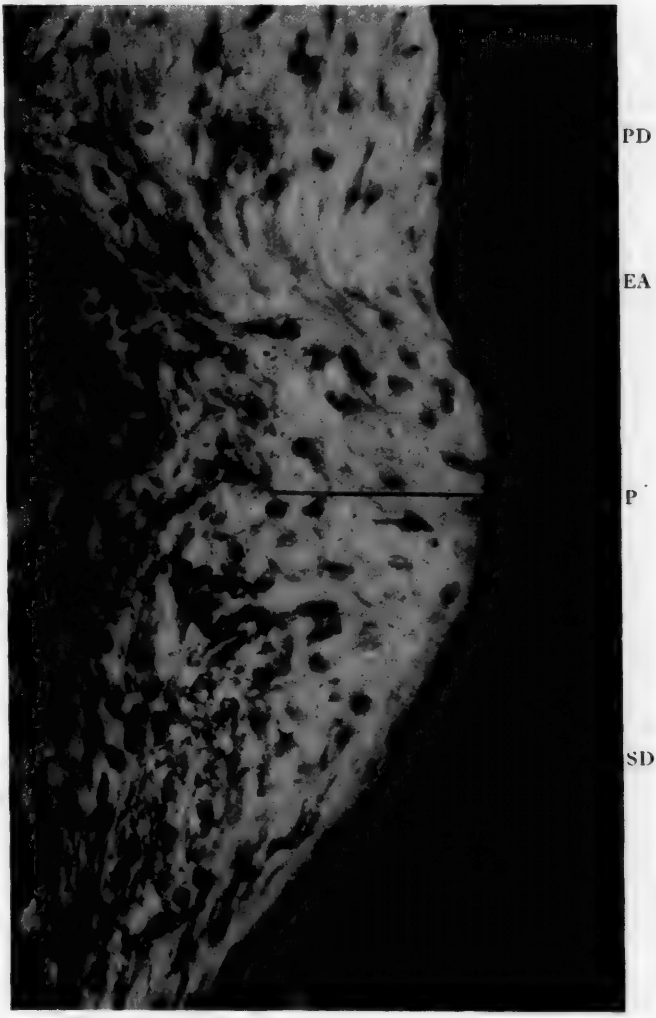

FIG. 6. HIGH MAGNIFICATION OF DEEPEST POINT OF EPITHELIAL ATTACHMENT IN ROOT CANAL

PD—Primary dentin. *EA*—Strand of epithelium attached to dentin and growing down into pulp canal. *SD*—Secondary dentin formed below point where epithelium ends. *P*—Pulp tissue, showing some enlarged capillaries.

dentin (*PD*), which forms the other wall of the pocket, is decayed and covered with debris and bacteria. It is probably this irritating condition of the rough dentin, similar to the action of calculus on the outer surface of a tooth, that caused the inflammatory condition of the pocket epithelium.

The epithelium extends, from the bottom of the pocket, down along the root canal in the form of an epithelial attachment. *Fig. 6* shows a high magnification of the deepest point in this epithelial attachment. The epithelium is attached to the primary dentin, and, at the deepest point of the attachment, begins the formation of new secondary dentin. In this way, the condition is similar to one sometimes found on the outside of the root when the epithelial attachment ends immediately above a fresh deposit of cementum—compare Stein (6) and Coolidge (1).

The order of change in the development of these conditions, although not recorded, appears to be: *first*, caries with consequent exposure of the pulp; *second*, pulpitis with a tendency to resist invasion by microörganisms. The result was a chronic inflammatory condition of the pulp with ulceration of the exposed surface. Caries finally destroyed the greater portion of the crown and an epithelial layer formed over the pulp tissue.

This epithelium probably developed in either of two ways. It might have been derived from the epithelial rests that in some instances are seen in the pulp tissue, as shown by the findings of Müller (4). In our case, the more logical way seems to have been by transplantation from the epithelium of the cheek or gums. The crown of the tooth was not broken down sufficiently to allow the pulp tissue to be in contact with the gum tissue. However, if epithelial cells loosened by friction in mastication were carried to the ulcerated surface of the pulp tissue, these minute grafts lived and finally developed there into this epithelial covering.

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PHYSIOLOGIC CHANGES IN THE DENTIN[1]

THEODORE B. BEUST, D.D.S., M.D., F.A.C.D.

School of Dentistry, University of Louisville, Louisville, Kentucky

In a paper read at the session of the American Dental Association, in Denver, July, 1930 (1), I submitted evidence that dentin undergoes physiologic hardening during the life of the tooth. This was shown to occur both in teeth traumatized by decay or abrasions, as well as in those apparently unaffected by such irritations. The increase in hardness was attributed to increased deposition of calcific matter. The part so modified was said to have become sclerosed. A distinction was made between sclerosed dentin that casts a shadow when being examined by transmitted light, and the similarly impregnated modification known as transparent dentin, the latter form being permeable to light rays. The first form of this dentin was referred to as "sclerosed opaque," the second as "sclerosed transparent," dentin. By means of a special technic sclerosed opaque and also sclerosed transparent dentin were shown to be of universal occurrence in mature teeth and their significance as factors in caries resistance was discussed. The object of this contribution is to comment on the phenomena upon which my views were based.

The material used in this investigation consisted of ground sections and of caries-free teeth that were halved longitudinally. The plane of the section in the latter case was through the pulp-cavity. Decalcified sections were not used. Formalin material, which had never been permitted to become dry, was selected. An exception to this rule was made where iodine had been used as a test (1). Differentiation between the normal or primitive form of dentin and the modified

[1] Read at meetings of the International Association for Dental Research, Hotel Peabody, Memphis, Tenn., March 21, 1931, and the Chicago Section, Hotel Stevens, Chicago, Ill., February 1, 1931. The work was supported in part by a grant from the Research Commission of the American Dental Association.

or sclerosed tissue was based largely on the staining reaction. In this differentiation the photographic camera, through which it was possible to render conspicuous changes not readily detectable by other methods, proved a valuable adjunct. In order to evaluate the specimens to be shown, it appears advisable to consider the technical procedures first.

The teeth were invariably stained in liberal amounts of a concentrated alcoholic stock solution of either basic fuchsin, Bismarck brown, violet black or nigrosin, diluted with an equal amount of water before use. For the sections, only fuchsin was used. Sections of about 0.75 mm. in thickness were exposed for at least twenty-four hours at 50°C., or seventy-two hours at room temperature. After the staining process, the sections were ground on both sides, to reduce them for examination. The time of exposure for halved teeth amounted to at least one week. This generally sufficed for differentiation. If this time appeared insufficient, the exposure was in many cases extended to a month. It is conceivable that over-staining may result in loss of the differentiation sought for. After being stained, the halved teeth were freed from superficial deposits by vigorous rubbing on a lubricated carborundum hone for about two minutes. The fuchsin appears to be the most practical, but the brown and black stains are best for photography. In view of the many ineffectual tests to determine the permeability of sclerosed tubules by sectional staining of dentin cross-sections, a reaming of the pulp cavity of the halved teeth for the purpose of removing secondary deposits was considered unessential, as these appeared to have no bearing on the objective. (Compare Fish—2.)

Fig. 1 shows the effects of the stain on teeth that have as yet undergone little modification. It is evident that the dentin in this state is permeable to stain. In contrast to this result, a section that has resisted staining is shown in *Fig. 2*. Only slight penetration in the neighborhood of the pulp cavity has occurred. For other specimens that have resisted staining see (1). The first demonstrable change in dentin sclerosis is indicated by the failure of the ground-substance (*fig. 3*) of the dentin to absorb stain. Young ground substance is at first permeable to dyes. Permeability, however, gradually diminishes until, at about the period of adolescence, it reaches a stage

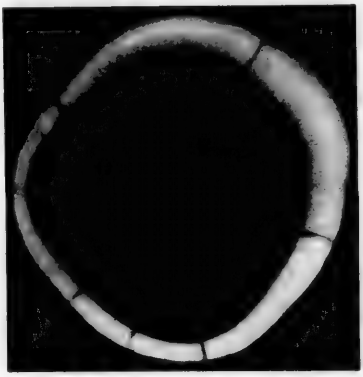

FIG. 1

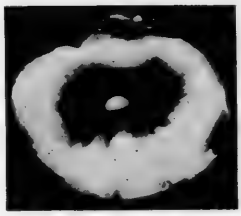

FIG. 2

FIG. 1. Cross-section of stained molar, showing penetration to amelo-dentinal junction. Reflected-light photograph.

FIG. 2. Reflected-light photograph of stained cross section from neck of tooth. Only slight penetration. Several months of exposure to stain.

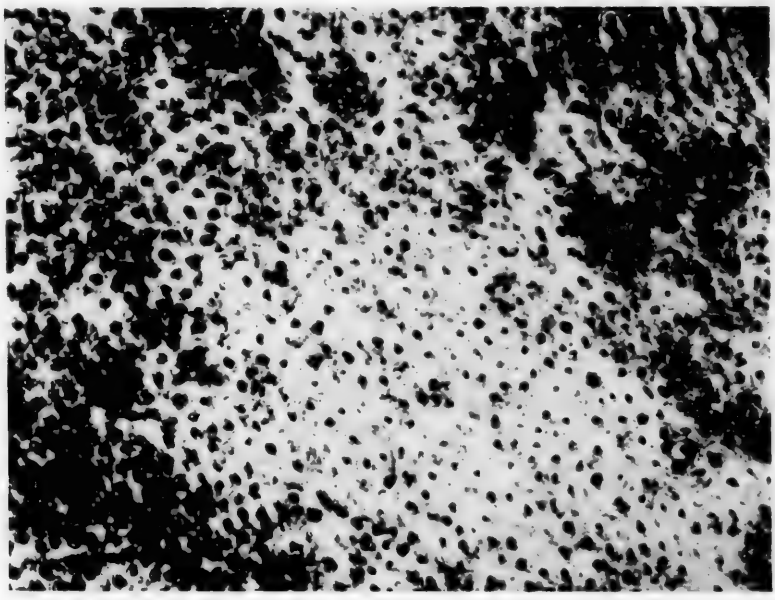

FIG. 3. Stained cross-section of dentin, showing cross-section of tubules and stained matrix (dark areas between tubules) in isolated areas. Transmitted-light photograph. Section stain.

where the peripheral basis-substance of the crown-apices of the anterior teeth begins to refuse stain. The dark spots on this photograph consist of stained tubules. The shadows in the ground substance represent areas where stains have entered it. The last part of the ground substance to stain is the Neumann sheath. The next noticeable modification occurs in the tubule itself. In some cases these contract to the point of complete obliteration, in others we find tubules that appear to be of normal diameter but have become impervious through deposition in the lumen.

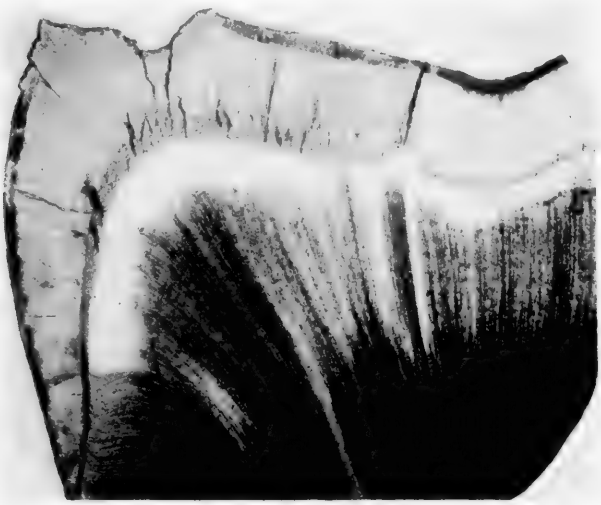

FIG. 4. Stained molar. No trace of caries. Periphery of dentin refuses stain. Sectional stain. Transmitted-light photograph.

It is evident that sclerosed tissue may, and usually does, alternate with permeable dentin. This gives rise to the peculiar fan-shaped effect caused by the distribution of the differentiated dentin (*fig. 4*). This appearance is explained, in sound teeth, by my conception of the physiologic elimination of the dental fibrils, as explained in my earlier paper (1). According to this concept the number of odontoblasts, together with their fibrils, are progressively decreased through normal contraction of the pulp cavity. Of such a numerical reduction there

can be no doubt. In view of the common occurrence of such sclerosed specimens as that shown in *fig. 2*, the only inference is that the eliminated fibrils calcify. They do not, in view of these experiments, persist as degenerated fibrils contained in open tubules. Seen in a longitudinal section of high magnification (*fig. 5*), stained fibrils, generally of reduced calibre. alternate with eliminated sclerosed tissue.

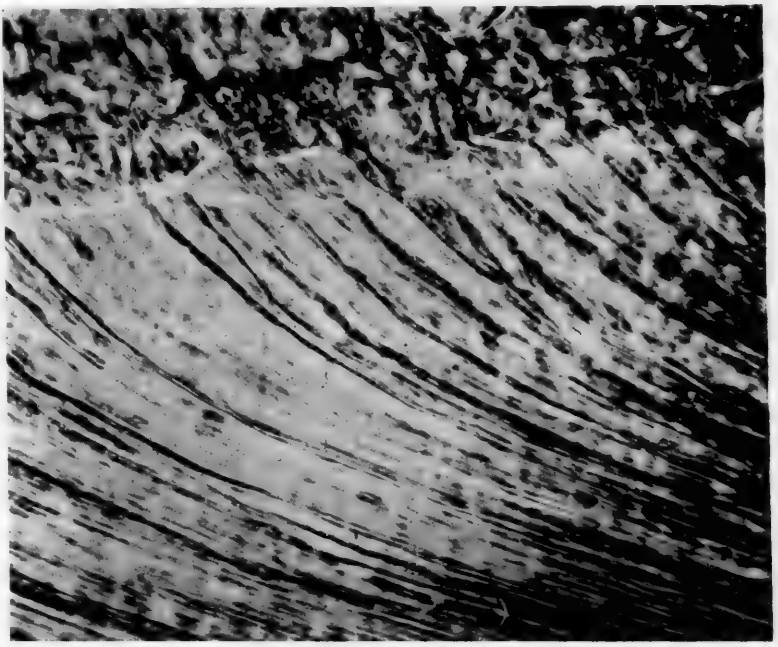

FIG. 5. Longitudinal section of dentin at periphery of crown. Sectional stain. Transmitted-light photograph.

This condition persists so long as the pulp retains its connection with the enamel. This it does, according to my observation, until the pulp disappears from the coronal part of the tooth.

Fig. 6 shows a photograph of a sectioned cuspid, taken by transmitted light. Under the abraded tip of this crown is shown an area of dentin of the sclerosed opaque variety. This, as will be observed, casts a shadow. The light spot in its depths is a small area of trans-

FIG. 6

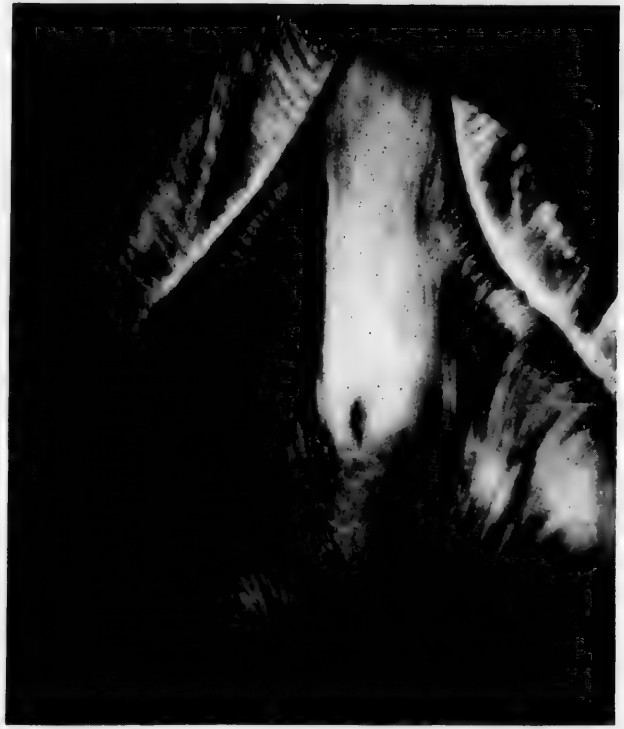

FIG. 7

FIG. 6. Stained abraded cuspid showing opaque sclerosis under morsal surface. Transmitted-light photograph

FIG. 7. Same specimen as that shown in *Fig. 6*, but photographed by reflected light. Opaque sclerosed areas appear white

parent dentin. Distributed throughout the remaining part of the crown are seen strands of opaque tissue. In order better to study this distribution, examine the section after it has been photographed on a black background (*fig. 7*). It will now be noted that the sclerosed opaque tissue is white, and that the tubules, being impervious, have failed to take up the stain. In my opinion the sclerosis of the dentin under the abrasion is a resistance area, physiologically formed to withstand the irritation caused by the impact of the opposing tooth; in other words, a reaction on the part of healthy fibrils. This sclerosed dentin is, I believe, identical in nature with normal growth changes, here hastened by irritation. (The word irritation is here used in the biologic sense.) While I relegate these changes to the realms of physiology, others prefer to regard them as pathologic. The views of Fish (2) concerning this question I have considered elsewhere (1). Today I would like to remark on a recent publication by Hatton (3). At this moment I have no right to doubt the accuracy of those who have detected recognized signs of degeneration in the fibrils or pulpal cells. This should be expected. Physiologic elimination of odontoblasts, together with their fibrils, unquestionably occurs. The disappearance of these structures must of necessity be accompanied by changes that may resemble, or be identical with, those observed in pathology. Yet I would like to register a difference. In pathologic irritation lime salts are mechanically precipitated from lymph that soaks into a dying organic matrix. Whereas in biologic irritation (synonymous with stimulation), there is a progressive deposition, an intussusception identical in nature with the process that converts dentin or bone from the consistence of childhood to that of maturity. In the first instance there is death; in the second, the end-result of normal growth.

I admit that we know too little about these tissues to make this question an issue today, but there exist valid objections to the idea that we are here dealing with pathology. Foremost in importance is the fact that sclerosis, as here defined, begins shortly after the normal eruption of the tooth, and increases progressively with advancing age in sound teeth. Sclerosis furthermore occurs in impacted teeth. This statement applies to all tissues of the tooth. We may find, in addition, further proof in the lower animals. For instance,

sclerosis of the fibrils, and consequent degeneration of the odontoblasts, normally occur so far from the morsal surface of the incisors of rodents that questions of pathology need not be considered. These facts singly and collectively indicate that we are here dealing with biologic changes induced by the provident and protective resources of the organism.

The manner in which the transition from the primitive to the mature state of the dentin progresses may be studied on almost any section of a sound tooth of an adult. *Fig. 8* shows a low power view of the

FIG. 8. Low-power view of specimen in *Fig. 4*, showing distribution of "sclerosed transparent" dentin.

specimen shown in part in *fig. 4*. It is evident that the stains have not traversed a sufficient number of tubules to darken the periphery of the dentin. This applies also to the root portion shown. Macroscopically the periphery appears to be, and barring isolated tubules is, made up of transparent dentin. Strands of sclerosed tissue extend from the enamel toward the pulp. Theoretically the time will arive when the strands will become wider, and more numerous, and finally confluent; the pulp will recede from the crown; the odontoblasts will become eliminated, and their fibrils sclerosed.

My conclusion is that teeth normally undergo a physiologic hardening in the manner described.

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THE LIPIN CONTENT OF DENTAL TISSUES IN RELATION TO DECAY[1]

A Preliminary Communication

CHARLES F. BÖDECKER, D.D.S., F.A.C.D.

Department of Oral Histology and Embryology, Columbia University Dental School, New York City

Dental decay is chiefly a disease of childhood and of youth. Its activity is usually greatly reduced after the age of twenty-five to thirty years according to the health of the individual. Miller's commonly accepted chemico-parasitic theory of dental decay offers no explanation for this and other clinical observations. Only if we accept the views of Bunting (3), Rodriguez (7), McIntosh and Barlow (6), and others, that dental decay is an infectious disease dependent upon the presence of specific organisms, can we comprehend such a change in the activity of this malady. With such a thesis we may assume that, after the age of twenty-five or thirty, oral conditions are such that the proliferations of the specific organisms of decay cannot develop as readily as in youth. This would result in the reduced activity of the disease.

That specific organisms, however, are not entirely responsible for this disease is shown by the following clinical observation. Decay is most common at the contact points of two closely adjoining teeth, and it is usual that *both* surfaces are attacked. This is what we would anticipate if we accepted the thesis that decay results from the bacterial fermentation of carbohydrate food lodged between the teeth. When, however, we encounter cases in which only one of the two closely contacting teeth decays, Miller's chemico-parasitic theory, *including* its modification of specific dental infection, does not hold.

[1] Presented at the ninth general meeting of the International Association for Dental Research, Hotel Peabody, Memphis, Tenn., March 21–22, 1931. Acknowledgment is hereby made of the financial assistance of the Commonwealth Fund of New York, in the pursuit of this study.

Such a case is shown in an x-ray (*fig. 1*) of a man aged 24. Here we note decay affecting the approximal surface of one of the maxillary incisors, while the other remains intact. If dental caries is caused solely by the retention of carbohydrate food that ferments under the action of aciduric organisms, *both* these closely adjoining surfaces should be affected. We must always keep in mind that it is not the bacteria but the acid which is supposed to attack the tooth; this dif-

FIG. 1. ONE-SIDED APPROXIMAL DECAY BETWEEN TWO MAXILLARY INCISORS OF MAN AGED 24

If dental caries is due solely to bacterial fermentation of carbohydrate food, *both* closely adjoining teeth should have decayed. As this has not occurred, it appears that in each tooth there exists some mechanism whose function inhibits or augments dental decay.

fuses through the entire food mass and mucous plaque. Some weakness of the affected tooth seems to be the only explanation for its decay. This lowered resistance to the acid may be explained in two ways: (*a*) structural weakness; or (*b*) pathological condition of the pulp.

(*a*) Improper diet may affect the calcification of the teeth during their formation, with consequent structural abnormalities of dentin and enamel. These would conduce to the rapid destruction of such

affected teeth if the carious process were active. But in the case shown in *fig. 1*, the carious and the non-carious teeth are pairs of incisors, developed at the same time. If nutritional disbalance were present during their formation, it would have affected both teeth and made them equally susceptible to dental decay. In this instance, therefore, the theory that faulty structure caused this unilateral approximal decay is untenable.

(*b*) The second possibility, that pathological conditions of the pulp were responsible for the decay, appears more plausible. The manner in which this operates will be briefly explained; it has been fully described elsewhere.

In order to comprehend the relation that the lipin content of the dental tissues bears to dental decay, it is necessary to recapitulate the theory of the author (2) concerning a probable predisposing cause of this disease. Percy R. Howe and the author believe that dental decay will finally be proved to be primarily a disease of the dental pulp, and that the health of this organ will be shown to be dependent on the health of the individual. It is further believed that the teeth have a protective mechanism which prevents their decay during youth under normal conditions. This mechanism is composed of the dental pulp whose odontoblasts secrete the dental lymph, which is transported through the dentinal "fibrils" to the organic matrix of the enamel. This protective mechanism of the teeth is particularly active during youth owing to the great permeability of the dental tissue at this time. Two or possibly three functions of the dental lymph are supposed to operate.

(A) *The dental lymph effects the post-eruptive calcification of the dentin and the enamel.* Newly erupted teeth do not appear to be as fully calcified as those that have been in function for some time. Some investigators who believe in a post-eruptive calcification of the dental tissues consider that it is achieved by the mineral salts in the saliva. This, the author believes, may occur, affecting, however, only the superficial zones of the enamel close to the surface of the tooth; the principal post-eruptive calcification probably takes place from the dental pulp.

(B) *During youth the buffer power of the dental lymph neutralizes the acid (lactic or malic) on the surface of the tooth.* The ability of the

dental lymph to fulfill this function is dependent on the presence in this fluid of some as yet unknown mineral salts. A similar function is fulfilled by the bicarbonate system of the blood in neutralizing sarcolactic acid.

(C) *The dental lymph may have a deterrent effect on the proliferation of aciduric organisms in the mouth.* This would result in reducing the exciting cause of dental decay.

The tenets of the above theory—the malnutritional theory as to the predisposing cause of dental decay—explain, for the first time, many of the puzzling clinical observations that could not be solved by other theories. One factor, however, gleaned from the study of pulpal conditions, puzzled the author for some time. According to the above theory, the presence of a sufficient quantity of dental lymph, having a proper constitution, seems essential in maintaining dental health. Observations of the dental pulp, however, in teeth of individuals of middle age, show that this organ has greatly reduced its activity at this time; that the odontoblasts tend to atrophy; and further that many of the dentinal tubules appear to contain fat. The dental lymph is greatly reduced in the teeth of individuals of middle age; yet this is the time that dental decay becomes less and less active. The explanation of this paradox may be found in this paper concerning the lipin content.

Before discussing the effect of the lipin content of the teeth and its relation to dental decay, it is necessary to consider the changes that occur in the pulp and cause a fatty metamorphosis of the dentinal fibrils. At the height of the activity of the dental pulp, the odontoblasts are of a large, cylindrical form. Their size ranges from 5 micra in width to 25 or 30 micra in length; they have well defined nuclei, situated at the base of the cell, farthest from the dentin they nourish. When the post-eruptive calcification of the dentin and enamel has been completed (this probably takes place approximately during the first ten years of adult life), the odontoblasts change their form from a columnar to a more cuboidal. Secondary dentin is laid down in the pulp chamber between the odontoblasts and the previously formed primary dentin, often as a result of the abrasion of the occlusal surfaces of the teeth. This cuts off innumerable dentinal fibrils from their vital connection, the odontoblasts. As a result of this severance, the

FIG. 2. PHOTOMICROGRAPH OF SECTION OF DECALCIFIED CARIOUS DENTIN, SHOWING FLOOR OF CAVITY ABOVE ×100

Specific stain (Sudan IV) shows fatty metamorphosis of dentinal fibrils leading toward pulp chamber below. Normal fibrils in their dentinal tubules are seen on either side.

dentinal fibrils, which now receive too little or no nourishment from the pulp, undergo a fatty metamorphosis.

Such changes have been shown to take place by Weber (8), Hatton (5), Euler and Meyer (4), and others. This subject has been much discussed during the last five years. The reports unanimously show the presence of traces of fat in the dentinal tubules affected by dental decay and abrasion. The number of papers which have been published during the last five years on this subject is indeed striking. The manner, however, in which the fat in the tissues may make the teeth immune to dental decay has not been understood; yet this may be of vital importance to a solution of the problem.

As stated above, for some years after the eruption of the teeth, decay, if it is at all active in the mouth, seems to progress with great rapidity. The rate at which dental destruction proceeds is in direct ratio to the constitution of the dental lymph and the permeability of the dental tissues. During this period a small superficial cavity may develop in a short time into a large one involving the dental pulp. Yet when healthy individuals reach the age of twenty-five or thirty, decay is either partly or wholly arrested. The question is how can this occur if the dental pulps have greatly reduced their activity, and but little dental lymph can permeate the tissues?

It is the purpose of this article to suggest that the lipin content of the enamel and dentin may be responsible for making these tissues impermeable and thus greatly reducing dental decay. These substances have not heretofore been considered as having a possible bearing on the immunity or susceptibility of the dental tissues to this disease. In fact very little is known concerning the fat present in the enamel and dentin. The chemical analyses of Von Bibra indicate that there is 0.2 percent of fat in enamel, and 0.4 percent in dentin. Weber, Hatton, Euler and Meyer have shown fat to be present in sections of carious dentin. These findings have been corroborated in our laboratory by staining this tissue with Sudan IV; the fat being defined as specifically red-colored masses filling the dentinal tubules (*fig. 2*). Under higher magnification, minute lipoid globules can also be noted in the matrix between these channels. Akamatsu (1) has reported fat in the enamel.

Investigators believe that a fatty metamorphosis of the dentinal

fibrils has taken place in those areas affected by dental decay. The difficulty that now comes up is to define the origin of the fat. According to older and now discarded theories, this condition developed as a result of a fatty degeneration of protoplasm, that is in this case, the dentinal fibril. New investigations, for example, those of the kidney, seem to indicate that fat is at all times a normal constituent of the tissues, and becomes visible only as a result of some pathological condition and the use of specific stains. Extending the premise that fatty metamorphosis of the dentin occurs as a result of dental decay (*fig. 2*), it is possible that fat is normally present in the healthy dental tissues. Little is as yet known concerning the chemical state of the invisible fat in the other tissues of the body, and nothing concerning that present in the teeth. If, however, an important physical characteristic of the invisible fat remains unchanged, that is, insolubility in water, there can be no doubt that the higher the percentage of fat present in the dental tissues, the lower would be their permeability to aqueous solutions. The lactic acid that is supposed to be the exciting cause of dental decay is in a watery solution; it could not attack a tooth whose fat content was comparatively high. In this connection, the recent and as yet unpublished report of Weston A. Price, on the beneficial results in reducing dental decay with diets containing ample amounts of butter, is indeed suggestive.

Qualitative and quantitative analyses of caries-immune and caries-susceptible teeth, as well as those in persons of various ages, are being made by Drs. Maxwell Karshan and Frances Krasnow; it will be interesting to note whether the fat content really varies in these different groups. Until we receive the results of these examinations and possibly those of similar ones made in other institutions, no conclusions can be drawn as to the value of the lipin content in the teeth. If it should be found that this content differs in the teeth of caries-immune and caries-susceptible individuals, we may be a step nearer the solution of this problem. Among results of such a report would be a further recognition of the nutritive value of fats, with possibly the more sane composition of dentifrices.

The author acknowledges with great pleasure the cheerful assistance and kindly advice given him in this work by Professors James W. Jobling, Alwin M. Pappenheimer and Maxwell Karshan.

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DENTAL INFECTIONS: COMPARATIVE SYSTEMIC EFFECTS OF QUANTITATIVE APICAL CHANGES[1]

A Study of 1503 Full-Mouth X-Rays

DANIEL E. ZISKIN, D.D.S.

School of Dental and Oral Surgery, Columbia University, New York City

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I. INTRODUCTION

During the last thirty years, the idea of focal infection has evolved from a theory into a method of practice. Today, if we are to judge from the medical and dental literature, this method of searching for and attempting to eliminate foci is universally practised. Yet, we are still confronted with this idea as a problem, for we find many conflicting opinions as to what constitutes a focus of infection, as to methods of discovery, and as to the importance and extent of eradicating these foci. Gruenberg pointed out some of these conflicts as follows:

(*a*) Bacteria are found in both healthy and pathological tissues.

(*b*) Pathogenic bacteria may be found in healthy as well as diseased individuals.

(*c*) While no direct connection has been established between apical infection and systemic disease, such inference may be drawn from the

[1] Read before the staff of the Department of Dentistry, Columbia University, January 21, 1931. Presented at the ninth general meeting of the International Association for Dental Research, Hotel Peabody, Memphis, Tenn., March 21–22, 1931.

many cases reported of people getting well as a result of extraction of infected teeth, and from the work of Rosenow on elective localization.

(*d*) Dental infection may be the result of some systemic disturbance rather than a cause of it.

(*e*) Altho it has been established that many people get well as a result of extraction, many do not.

(*f*) While some investigators have reported cures in cases of insanity, or other diseases, by extractions, other investigators have reported cures in similar types of cases by an entirely different method, such as psychoanalysis.

(*g*) Admitting the possibility of the focal infection theory, it does not follow that all infected teeth must be removed or that focal infection is the only source of trouble.

(*h*) Is it possible to retain infected teeth for a long period of time safely, thus adding to comfort of the masticating apparatus, or would such a procedure endanger the future health and chance for recovery if systemic disease did develop?

How are we going to reconcile all these conflicting views, and attempt to conduct ourselves sanely in routine practice? Shall we adopt the attitude of the "whites," and join the ranks of those holding that all pulpless teeth should be extracted? We would indeed be spared a great deal of confusion, and find diagnosis simple. Or shall we adopt the attitude of the "blacks," and resort to the practice of treating and retaining all infected teeth. This procedure would also simplify diagnosis and render practice easy. If neither "black" nor "white," shall we assume the attitude of the "greys" and extract some pulpless teeth and retain others? But here we encounter a series of complications. *First:* we may be accused of lacking sufficient knowledge to take a stand, and thus be unfit to render proper service to our patients. For if we do not extract *all* pulpless teeth, we may retain some that are a menace; and if we extract *some* pulpless teeth, we may be depriving the patient of useful organs that could be retained for long periods of time. *Second:* how are we to know which teeth to extract? Will we be guided by radiographic findings, by clinical findings (that is, swelling, pain, tenderness on percussion, fistulae, or pulp testing), blood analysis (that is, counting, culture, chemical, bacterial), other chemical analysis, systemic condition,

family history, age, susceptibility, or combinations of the foregoing? If we are to rely largely on radiographic findings, what signs are we to follow? Shall we depend on quantitative reactions (that is, large rarefactions, small rarefactions, or negative areas)? Which is the most harmful, the large area or the negative? Shall we depend on qualitative reactions (that is, rarefaction of bone, condensation of bone, rarefaction surrounded by a zone of condensation); or shall we assume that x-rays are unreliable, because they show only half, at most, of the bone surrounding a tooth; or shall we attempt to be clairvoyant in determining what the other half might disclose?

Several years ago, I began collecting data with the purpose of attempting to clarify some of these perplexing problems. At that time, I was impressed with the doctrine of Price of Cleveland, and hoped that he was correct in his conclusions, for he stated: "When the diagnostician thoroughly visualizes this great new truth, he will find himself playing the game of diagnosis with marked cards. He can pretty nearly make out the patient's chart from the family record, or vice-versa." Though I know it is poor sportsmanship to play a game with marked cards, I was perfectly willing to play the game of dental diagnosis in this way. However, on considering the great volume of literature published by both the medical and dental professions after the utterance of Price's "great new Truth"—a few accepting and the large majority doubting his views—we must pause and reflect that dental diagnosis is still open to question.

II. GENERAL REVIEW AND SUMMARY OF THE LITERATURE

A complete review of the literature would be time-consuming. Therefore, I shall give below only a composite of the views of some of the outstanding men in the dental and medical fields in order to broaden our basis for judgment.

I. In patients who have *secondary disease* that may be attributed to focal infection, all pulpless teeth should be extracted.—*Thoma.*

II. In patients who are *well*, "all dead teeth" should not be condemned.—*Thoma.*

III. That bacteria within dental foci have an elective affinity for organs and tissues similar to those in the patients is demonstrated by

intravenous injections in rabbits, and the production of chronic dental foci in dogs.—*Rosenow, Meisser.*

IV. The mere presence of bacteria in a pulpless tooth, or in the tissue about it, cannot be regarded as sufficient evidence that the body is being injured by such bacteria or even menaced by them. On the contrary, such bacteria may be of advantage in maintaining and increasing the powers of the normal defensive mechanisms of the body. The specificity of the bacteria involved has not been proved, and the evidence favoring the theory of elective localization is so open to misinterpretation, and so limited in its practical application, that it cannot be considered as a help in the solution of the problem. A certain general bacterial adaptation to environment is accepted by everyone, but the factors on the side of the host are more variable and are far more important.—*Barker, Holman.*

V. The arguments that not all pulpless teeth are infected or give rise to serious systemic disease put forth by some for the retention of pulpless teeth, are similar to those brought forward some years ago against the idea of water-born typhoid. Only a small proportion of the population that consumed the polluted water contracted typhoid fever, and therefore the source in those that did must be other than the water.—*Rosenow.*

VI. We have no right, as yet, to assume that the removal of mouth infection is going to exercise a profound influence upon the incidence of the degenerative diseases of the latter decades of life.—*Hatton.*

VII. Persons who have infected teeth remain in apparently good health for years, but so do people afflicted with alcoholism, gallstones, tuberculosis, arteriosclerosis, diabetes, Bright's disease, valvular disease of the heart and many other diseases which cause symptoms when it is too late to cure them.—*Nodine.*

VIII. Patients with apical abscesses, and systemic disease apparently due to focal infection, should not have any useful, even though infected, teeth drawn until other foci have been investigated, and eradicated. The gravity of the systemic infection must be weighed against the handicap involved in the loss of useful teeth.—*Taussig.*

IX. Clinicians are disappointed in the results obtained by the removal of foci of infection in the treatment of diseases, even of

proven metastatic origin. The lesson to be learned from the demonstration of a definite relation of foci of infection to various systemic diseases is the need for the removal of such foci before metastasis to other organs has taken place. Disease, once initiated, is often not affected by the removal of the cause.—*Haden.*

X. The presence of bacteria does not mean infection, and only becomes such when the resistance is lowered and the tissue overwhelmed. Incidental bacteria found at the root ends of pulpless teeth, or even teeth with vital pulps, which do not produce any local cellular reaction, are likely to be harmless, either locally or constitutionally.—*Chamberlain, Moorehead.*

XI. Since nearly all pulpless teeth are infected, it is a safer procedure to cut down on the amount of streptococci feeding into the system.—*Dunning, Blum.*

XII. Researches carried out on different groups of patients, that is, a hospital group or a group from dental practice, will give different results.—*Rickert.*

XIII. Chronic periapical infections may either be associated with condensation of bone and encapsulation, or with absorption and localized extension of the infection.—*Hatton.*

XIV. The most obvious and important conclusion from the research work hitherto accomplished is that root-canal operations should be done with much more care and thoroughness than has ever been the case with a large majority of practitioners.—*Noyes.*

XV. Probably the greatest fallacy in determining the presence of oral foci of infection is that of pronouncing as safe, or harmless, the pulpless tooth that shows no periapical destruction. Teeth with perfect root-canal fillings, which were placed in sterile manner by experts, after the scrapings from the root canals were proven sterile, and which had been watched and remained symptomless and x-ray negative, were extracted and from some of these were isolated streptococci that produced marked lesions in animals, in tissues corresponding to those affected in patients.—*Moll, Rosenow, Haden.*

XVI. Dental foci are more often contributing rather than initiating factors to other infective involvements. They may aid in establishing conditions due to a metabolic disturbance or add to their intensity by means of what we may call sensitization.—*Rickert, Libman.*

XVII. If a result is obtained by removing one or more teeth, the effect may have been obtained by the occurrence of a febrile reaction and not because the infected teeth were causal.—*Libman.*

XVIII. The root of a pulpless tooth merely furnishes a place of lowered resistance and becomes infected secondarily in the same way as does the heart and kidney.—*Solis-Cohen.*

XIX. Purulent metastasis in remote parts of the body arising from dental infections are very rare.—*Shottmuller.*

XX. The mere presence of an area of chronic sepsis coincident with diseases elsewhere cannot always be taken as proof of cause and effect. There is more than a possibility that both the focus and disease may be caused by the same condition, and if their cause be searched for in their endocrine autonomic apparatus, they can be given relief more successfully and with less heroic measures.—*Broderick.*

XXI. A tooth of the guinea pig can act as a channel for the introduction of an antigen to the general circulation, resulting in antibody formation, with consequent hypersusceptibility in remote tissues.—*Weisberger.*

Since it has been demonstrated that most pulpless teeth are infected, and that infection sets in even in cases of treatment by experts, these teeth are considered a menace to the health of the individual and should be removed. Many observers support this contention, as well as the one that dental infections may be secondary, thus establishing the fact that dental infections as well as others may be metastatic. In addition, the numerous cases in which patients are reported as having been benefited by the removal of infected teeth, give further support to the focal-infection theory. The work of Rosenow and his associates as to elective localization, while questioned by many, has never been disproved, but on the contrary has been verified by some, and therefore cannot be ignored. The most recent and perhaps the strongest contention in favor of the focal-infection theory is the idea that infected teeth may cause allergic reactions.

Of the contentions tending to detract from the focal-infection theory, perhaps the strongest is the fact that, in some cases where improvement is noted after the elimination of infected teeth, this improvement may be due to a febrile reaction caused by the extraction rather than the extraction itself. The fact that other foci may be

present tends to minimize the importance of a dental focus, and gives rise to the idea that the general bodily resistance must first be lowered before the dental focus can become harmful. A rather interesting view is advanced in the contention that dental infections, since they are low grade, may be instrumental in establishing an immunity against the more serious complications that are usually attributed to focal infections. That disturbances in the endocrine autonomic apparatus may be responsible for both the systemic disease and the dental infection has also been suggested.

In deciding which teeth to extract, and the extent of this procedure, consideration must be given to the gravity of the systemic disease, and the age of the patient. Whether a cellular reaction has occurred about a pulpless tooth in question is a criterion to be used in determining the suitability of retaining the tooth. Strategic teeth should be allowed to remain. Since negative teeth are not safe, and since chronic infections about the teeth may result in either condensation or rarefaction of bone as noted in the x-ray, and since it is advisable to reduce the number of streptococci in the body, the removal of all pulpless teeth has been advised.

III. AUTHOR'S STUDY

A. General procedure

My study is made up of a group of 1503 patients who attended the dental clinic at the Minneapolis General Hospital. These patients compare favorably with those coming into the Dental School here. They consisted of (*a*) patients referred from the medical clinics for the removal of dental foci of infection, (*b*) patients referred from the wards of the hospital for the same purpose, (*c*) patients referred from various social agencies, solely for charitable dental treatment. The routine practice was to take full-mouth x-rays in all cases. The x-rays were read by me and cross-filed. They disclosed a group showing large areas of bone rarefaction, a second group showing small areas, a third group showing negative areas, a fourth group showing both large and small areas, and a fifth group showing bone condensation. The latter group, however, was so small that I did not include it in this study. My cross-file also contained a group which had no devitalized

or pulpless teeth. This group was used as a control. Another group showed pulpless teeth but no systemic findings. The latter group I used as a second control. The medical diagnosis in this study was taken from the patient's hospital record.

The purpose of the study was two-fold: (*a*) to prove or disprove Price's findings as related to x-ray interpretation, and (*b*) to see whether there is any means by which we can correlate dental findings with systemic diseases, and draw conclusions from this correlation that will aid in dental diagnosis. Dentistry of the past taught that the size of the area of rarefaction determined the extent of pathology about a pulpless tooth. Though this idea was modified from time to time by various men, as may be noted in my review of the literature, the prevailing method of practice is to rely mainly on what the x-ray shows. Attempts have been made to use other indices in determining dental infections. Most noteworthy among these is the work of Torren, who in 1922 published the result of his work dealing with diagnosis of oral infections by blood examinations. This work, however, was shown by Epstein and others to be erroneous. Other methods likewise proved to be inadequate when, in 1922, Price made the startling claims published in his monograph. Since that time, Price's work has been largely discredited by Hartzell, Rickert, Barker, Hatton, and others. Nevertheless, many men are still influenced by Price's claims as to radiographic interpretation, which are quoted in part below from the volume in question:

"The local and systemic expressions are not only related, but are both symptoms of the same controling forces and conditions" (p. 120).

"It may be, and frequently is, true that the patient suffering severely from a systemic reaction caused by a dental infection, shows very little absorption compared with that which the same dental infection would produce in a patient with ample and high resistance" (p. 176).

"Since an adequately active defense against a dental infection produces a vigorous local reaction with attending extensive absorption, and the products of inflammatory reaction—namely, exudate and plasma in sufficient quantity to require an overflow, usually spoken of as pus from a fistula—this overflow may be, and usually is, evidence of an active defense and is constituted almost wholly of neutralized products, and is often sterile; and such a condition is much more safe than the same infected tooth without such an active local reaction" (p. 183).

"The structural changes which develop about infected teeth are expressions, primarily, of the type of reaction of the host to this irritant and, as such, are therefore, when understood, a direct means for interpreting that individual's defensive mechanism" (p. 580).

These findings tend to reverse entirely our ideas about dental diagnosis, for those teeth that we previously believed to be the least harmful, Price considers the most harmful, and vice-versa.

In my study I approached the problem from a different angle. I studied the x-rays first, and arranged them into the various groups, and later obtained the medical diagnoses. While many of these diagnoses were not sufficiently detailed, they were adequate for the purpose of this study.

B. Groups of patients and clinical data

The x-rays were arranged into the following groups:

Group I. Those showing large areas of rarefaction.

Group II. Those showing small areas of rarefaction.

Group III. Those showing no areas of rarefaction.

Group IV. Those showing combinations of groups I, II, and III.

Group V. Those showing no pulpless teeth. This group is used as a control.

The medical diagnoses (of patients) were arranged into the following groups:

Dental.—Patients who had no apparent systemic complaints, but had pulpless teeth. They came to the clinic for dental attention only. This group constitutes a second control.

Plus.—Patients classified as having systemic diseases that may be considered as of focal-infective origin.

Minus.—Patients classified as having systemic diseases that were definitely of non-focal-infective origin.

Plus-minus.—Patients classified as having systemic diseases that may or may not be attributed to focal infection. The medical diagnoses were made by physicians. In the classification and segregation of the list of systemic diseases into the groupings, I was assisted by a physician, Dr. David D. Moore, Instructor in the Department of Medicine, and Assistant Physician, Presbyterian Hospital, Medical

TABLE 1

Comparative systemic effects of quantitative apical changes: 1503 full-mouth x-rays

DIAGNOSIS	LARGE RAREFIED AREA: GROUP 1	SMALL RAREFIED AREA: GROUP 2	NO APPARENT RAREFACTION: GROUP 3	LARGE-SMALL AND NO RAREFACTION: GROUP 4	TOTAL OF GROUPS: 1, 2, 3, 4	NO PULPLESS TEETH PRESENT: CONTROL: GROUP 5	TOTAL
No apparent systemic complaint: Dental Control	255 55.31%↑ 43.00%←	188 52.95%↑ 31.70%←	105 51.98%↑ 17.70%←	45 56.25%↑ 7.55%←	593 54. %↑ 74.59%←	202 49.87%↑ 25.40%←	795 52.89%↓
Possible focal-infective origin +	21 10.19%↑ 33.87%←	22 13.11%↑ 35.48%←	13 13.40%↑ 20.96%←	6 17.14%↑ 9.67%←	62 12.27%↑ 71.26%←	25 12.31%↑ 28.73%←	87 12.28%↓
Definite non-focal-infective origin −	136 66.01%↑ 40.47%←	108 64.67%↑ 32.11%←	66 68.04%↑ 19.52%←	26 74.14%↑ 7.73%←	336 66.53%↑ 74.5 %←	115 56.64%↑ 25.49%←	451 63.7 %↓
Possible focal or non-focal-infective origin ±	49 23.78%↑ 45.79%←	37 22.15%↑ 35.57%←	18 18.55%↑ 16.82%←	3 8.57%↑ 2.80%←	107 21.18%↑ 62.94%←	63 31.02%↑ 37.05%←	170 24.01%↓
Total +−± Groups	206 44.69%↑ 40.79%←	167 47.04%↑ 33.06%←	97 48.01%↑ 19.20%←	35 43.75%↑ 6.93%←	505 45.99%↑ 71.32%←	203 50.12%↑ 28.67%←	708 47.1 % ↓
Total	461→ 41.07%	355→ 32.33%	202→ 18.39%	80→ 7.28%	1,098 73.05%→	405 26.94%→	1503

Center—Lecturer and Demonstrator to the dental students on the subject of clinical medicine. *Table 1* shows the arrangement of the cases in the various groups and the percentages.

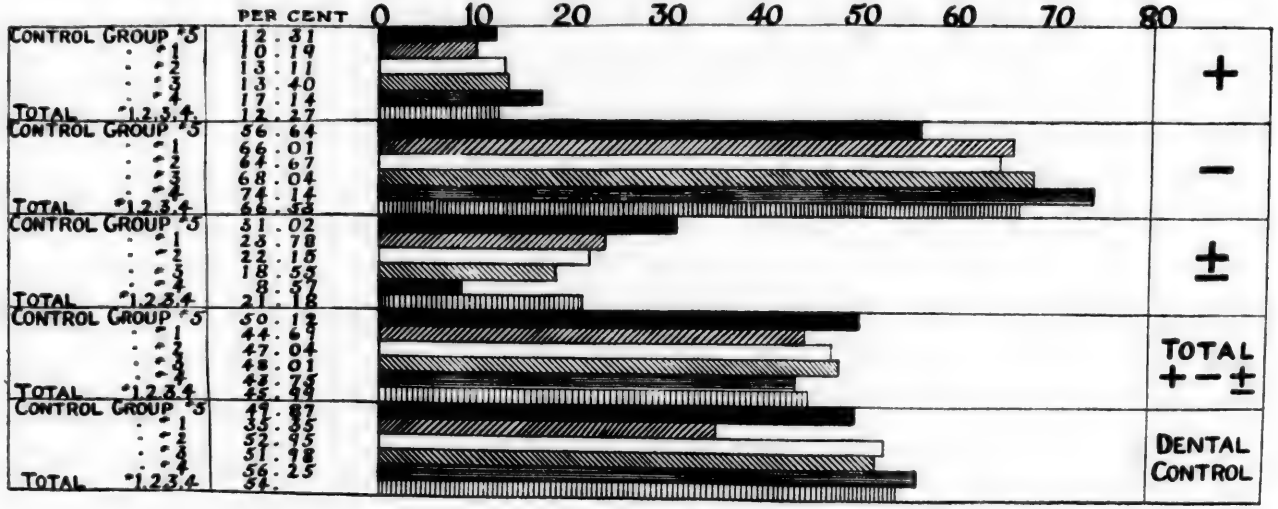
The importance of the findings in *Table 1* is not the actual number of cases in each group, but the comparison of the groups as shown by the percentages, which are given for both the medical diagnoses, and the x-ray findings. The Dental Group is used as a control for the medical diagnoses; Group 5, as a control for the x-ray findings. To illustrate this point further, there were twenty-one cases whose medical diagnoses placed them in the Plus Group, who also showed large rarefied areas about the pulpless teeth in the x-rays. Twenty-one is 10.19 per cent of Group 1, and 33.87 per cent of the Plus Group. Totals are shown first, excluding the control groups, and then including the control groups.

Graph 1 is a bar diagram showing the comparison of the percentages of Groups 1, 2, 3, 4, 5, and totals. *Graph 2* shows a comparison of the percentages of the Dental, Plus, Minus, Plus-minus groups, and totals. *Table 2* shows the average age for each group represented in *Table 1*.

C. Discussion

We are interested in this study to ascertain whether the various types of apical involvement occur consistently in any groups of metastatic involvements. In other words, is there any connection between large rarefied areas, as noted in the x-rays of pulpless teeth, and freedom from degenerative diseases; and between negative findings about the apices of pulpless teeth, and susceptibility to degenerative diseases. Price says that a definite relationship does exist. My findings indicate that there is none for, when the percentages of the various groups are compared, they are found to be strikingly similar. That is to say, persons having degenerative diseases and also pulpless teeth show a comparable distribution of the large, small, and negative areas of rarefaction about their pulpless teeth. Those persons having diseases that are definitely non-focal in origin also have almost the same proportions of large, small, and negative areas of rarefaction about their pulpless teeth. And, in the control group, having no systemic complaints, the same is true; namely, there are practically the same proportions of large, small, and negative areas of rarefaction.

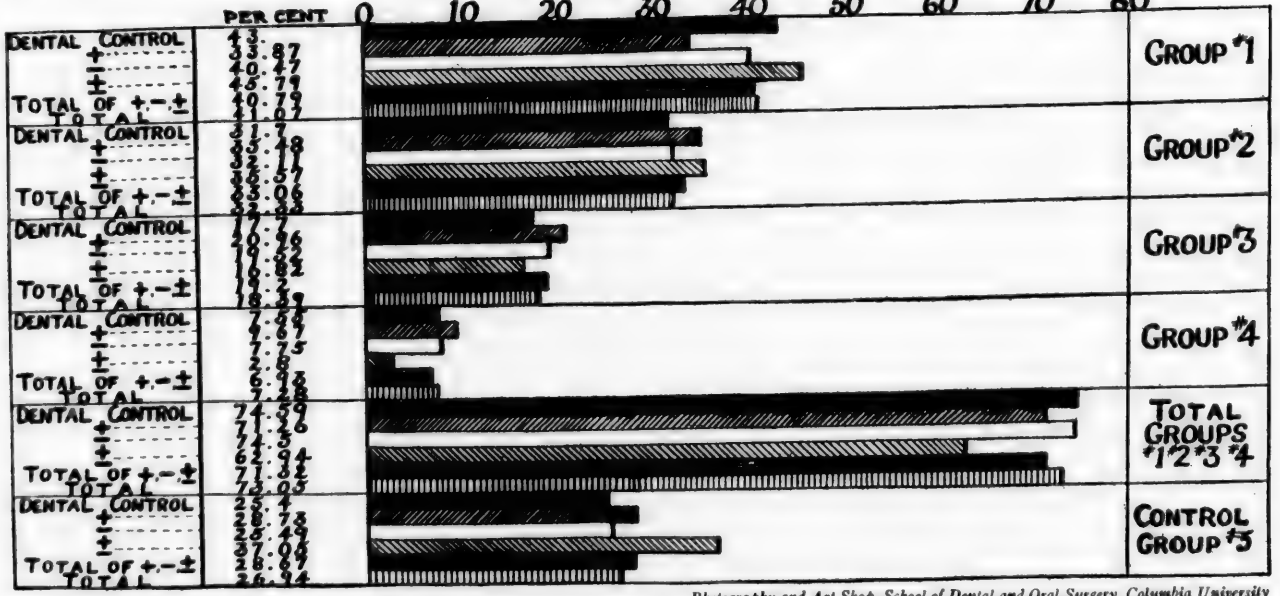
PERCENTAGES SHOWING
SYSTEMIC INVOLVEMENTS IN RELATION TO APICAL INFECTIONS
COMPARISON OF QUANTITATIVE APICAL CHANGE GROUPS



Photography and Art Shop, School of Dental and Oral Surgery, Columbia University

GRAPH 1

PERCENTAGES SHOWING
SYSTEMIC INVOLVEMENTS IN RELATION TO APICAL INFECTIONS
COMPARISON OF VARIOUS DISEASE GROUPS



Photography and Art Shop, School of Dental and Oral Surgery, Columbia University

GRAPH 2

If we try to find how the group that has large areas of rarefaction about the pulpless teeth is distributed among the Plus, Minus, and Plus-minus groups, we discover that the variation is so slight as to have no significance. The same is true of the small- and negative-area

TABLE 2

Average age for each group represented in Table 1

DIAGNOSIS	GROUP 1	GROUP 2	GROUP 3	GROUP 4	TOTAL OF GROUPS: 1, 2, 3, 4	GROUP 5	TOTAL
Dental Control	38.29	34.09	34.4	37.	35.94 ←	24.64	High 78 Low 9 Avge. 33.68 ←
+	45.52	40.95	33.38	37.	39.21 ←	35.69	High 84 Low 18 Avge. 38.5 ←
−	37.02	35.75	35.51	37.92	36.55 ←	30.57	High 76 Low 13 Avge. 35.35 ←
±	40.57	37.50	35.87	41.5	38.86 ←	29.95	High 76 Low 8 Avge. 37.07 ←
Total + − ±	↑ 41.03	↑ 38.06	↑ 34.92	↑ 38.8	↑ 38.2 ←	↑ 32.07	High 77 Low 13↑ Avge. 36.97 ←
Total	↑ 40.35	↑ 37.07	↑ 34.79	↑ 38.35	↑ 37.64 ←	↑ 30.21	High 84 Low 8↑ Avge. 36.15 ←

groups. The control group, which has no pulpless teeth, is distributed in a manner similar to the other groups. From this latter finding, we might be led to infer that, since patients with no pulpless teeth have diseases similar to those with pulpless teeth, the pulpless teeth are not a factor in the production of the disease. There are two things

that we must consider here as bearing on this hypothesis. *First:* I did not take into consideration missing teeth, nor other types of mouth infections such as pyorrhea. *Second:* I did not make a study of other foci of infection, such as tonsils, sinuses, etc., in their relation to the systemic disease, or to the dental focus. As I stated before, my purpose was to find whether there was any relationship between the size of the areas of rarefaction and the systemic disease. Also, whether there was any constancy in my findings to indicate which type of apical change occurs most frequently in connection with systemic disease, so that we could determine which was the most harmful.

In this connection I call attention to two similar studies, in which the findings were comparable with mine. Dr. Lee K. Frankel, of the Metropolitan Life Insurance Co., found, in a study of 19,000 life insurance examinations, that men with "heavy dentistry" had no more systemic disturbances than men without "heavy dentistry." By "heavy dentistry," he means a great deal of restorative work. He draws the conclusion from his findings that the dental profession has not been careful enough to reserve acceptance of novel statements and new methods until they have gone through the fire of the most thorough investigation and research. His findings are given in *Table 3.*

Dr. Edward C. Stafne, of the dental staff at the Mayo Clinic, studied a group of 1500 patients. Of these, 500 were diagnosed as having infectious arthritis; 500, duodenal ulcers; and the remaining 500, who were patients coming in for examinations at the Mayo Clinic some of whom had the foregoing findings, but most of whom did not, were used as a control group. His findings are given in *Table 4.* His study was made of the x-ray findings on these patients, and he considered the large areas of rarefaction and negative areas, as I did. At a first glance it would seem that, since his *Part 1* shows a much larger number of pulpless negative teeth than pulpless teeth having areas of rarefaction (in the Arthritis Group, as compared with the other two groups), his findings are in agreement with those of Dr. Price. If, however, the difference in the number of missing teeth between the Arthritis Group and the control group is added to the Arthritis Group (in the column showing teeth having rarefied areas), then we find that his two groups (pulpless teeth negative, and pulpless teeth with

rarefaction) are nearly equal. In order to get a true picture this should be done, for, as he states, the arthritic patients have had more treatment before coming to the Mayo Clinic than the other groups.

TABLE 3

*A consideration of evidences on oral sepsis in relation to systemic disease**

Percentage of cases showing specified impairment in two groups: (a) 6960 white males having "heavy dentistry" and suspected dental infection; (b) 9702 white males not showing "heavy dentistry" and suspected infection.

IMPAIRMENT	PERCENTAGE SHOWING SPECIFIED IMPAIRMENT	
	6960 white males with "heavy dentistry"	9702 white males not having "heavy dentistry"
Total cases	100.0	100.0
Enlarged, septic or buried tonsils	28.9	24.3
Functional heart murmur or irregularity	6.6	5.6
Mitral murmur—stenosis	.2	.1
Mitral murmur—regurgitation	.6	.8
Slow pulse, below 58	1.4	1.3
Rapid pulse, above 90	8.7	8.0
Arterial thickening; slight	17.7	11.8
Arterial thickening; moderate	5.6	3.9
Arterial thickening; marked	.7	.6
Blood pressure: 15–25 below average for age	10.0	9.7
Blood pressure: 25 or more below average for age	2.5	2.2
Blood pressure: 20–40 above average for age	6.3	6.1
Blood pressure: 40–60 above average for age	.9	.6
Blood pressure: 60 or more above average for age	.3	.3
Acid stomach	10.3	9.5
Gastric disturbances	7.9	8.4
Tenderness in region of appendix	2.5	2.2
Dizziness (vertigo)	6.7	5.9
Normal urine	73.1	74.8
Albumin: slight trace	13.1	11.6
Albumin: definite trace	2.5	2.2
Albumin: marked amount	.8	.7
Sugar: trace	3.5	3.4
Sugar: marked amount (quantitative)	.4	.5
Pus (large amount or persistent numerous leukocytes)	2.9	2.6
Casts: granular or epithelial	.5	.2
Casts: hyaline	4.1	3.8

* Lee K. Frankel, Ph.D., New York City, N. Y., Third Vice-President, Metropolitan Life Insurance Co., *Dental Cosmos*, 1924, lxvi, p. 38.

A large part of this treatment consisted in the extraction of infected teeth. I think we are justified in assuming that the extracted teeth were those considered by dentists to be the most harmful—the ones having the large rarefied areas. We are justified in this conclusion because, where teeth were extracted, pulpless-negative teeth were allowed to remain, and more teeth were missing in the arthritic group

TABLE 4

Dental conditions found in duodenal ulcer and arthritis[1]

Part 1 (Table I): Pulpless teeth

GROUP[2]	ABSENCE OF PULPLESS TEETH AND PERIAPICAL INFECTION		PRESENCE OF PULPLESS TEETH							
					Resorption around apex				Condensation around apex	
	Patients	Per cent	Patients	Teeth	Patients	Per cent	Teeth	Per cent	Teeth	Per cent
Arthritis	280	56.0	220	400	31	14.2	127	32	24	6.0
Duodenal ulcer	151	30.2	349	887	149	42.8	673	75	9	1.0
Control	132	26.4	368	1013	162	44.0	689	68	35	3.5

Part 2 (Table III): Loss of teeth

GROUP	MISSING TEETH		EDENTULOUS PATIENTS	
	Average number missing for each patient	Per cent	Number	Per cent
Arthritis	11.9	37.2	86	17.2
Duodenal ulcer	8.2	25.6	10	2.0
Control	7.6	24.0	13	2.6

[1] Edward C. Stafne, D.D.S., Section on Dental Surgery, Mayo Clinic, Rochester, Minn., *Dental Cosmos*, 1929, lxxi, p. 404; April.

[2] Five hundred patients examined in each group.

than in the others. Hence, Stafne states that his findings are not in agreement with Price's.

IV. SUMMARY AND CONCLUSIONS

In this study 47.10 per cent of the patients were sick, 52.89 per cent were well. Of the sick, 71.32 per cent had pulpless teeth, 28.67 per cent had not.

Of those who were apparently *well*, 74.57 per cent had pulpless teeth, 25.4 per cent had not. Of those who had pulpless teeth, 45.99 per cent were *sick*, 54 per cent were *well*. Of those who had no pulpless teeth, 50.12 per cent were *sick*, 49.87 per cent were *well*. Since our comparisons in these instances are so nearly alike, we cannot draw any conclusions, either in favor of or against pulpless teeth, as related to systemic disease.

Of those who were *sick*, who had pulpless teeth, we find comparable percentages of the large, small, and negative rarefied areas as noted in the x-ray. Of those who were *well* and had pulpless teeth, we find comparable percentages of large, small, and negative rarefied areas as noted in the x-ray.

Of those who had pulpless teeth showing *large* rarefied areas in the x-ray, we find comparable percentages of the various types of systemic conditions (persons without systemic complaints, diseases that are possibly of focal-infective origin, diseases that are definitely non-focal, diseases that may be either focal or non-focal). The same is true of those groups in which the pulpless teeth show *small*, negative, or "mixed" rarefied areas.

From these findings we must conclude that no more importance can be attached to the large rarefied areas as noted in the x-ray, than to the small or negative areas, and vice-versa.

The *clinical significance* of these findings may be summed up as follows:

Since this study neither proves nor disproves the focal-infection theory, *we must be guided by other studies in forming our judgments concerning the relationship between pulpless teeth and systemic disease.*

If we are of the opinion that pulpless teeth are a factor in the production of systemic disease, and we decide upon extraction, then we must extract all of them, for as yet we have no means of determining which are safe and which are not. We can modify this attitude only in cases in which it would be obvious that the "cure" would be worse than the "disease." In such cases, only strategic teeth should remain regardless of the x-ray findings as to large areas, etc.

Interpretations of the dental x-ray prove inadequate in finally determining those teeth that should be extracted and those that should be retained.

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FURTHER COMMENT ON THE AMERICAN CHILD HEALTH ASSOCIATION'S MONOGRAPH III ENTITLED: "PUBLIC HEALTH ASPECTS OF DENTAL DECAY IN CHILDREN"

ORAL HYGIENE COMMITTEE OF GREATER NEW YORK[1]

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I. INTRODUCTION

The last three numbers of the *Journal of Dental Research* contained a succession of papers on the quality of the American Child Health Association's Research Monograph Number III.[2] The present statement is a direct analysis of the complete reply to our original protest. For convenience in discussion, the response by Dr. Palmer and his associates is referred to here as "Dr. Palmer's reply."

The conditions that led us to publish our original protest were indicated early therein by this statement (p. 502): "It is deeply gratifying

[1] Sub-committee on the American Child Health Association's Research Monograph III; H. Shirley Dwyer, Thaddeus P. Hyatt, William J. Gies, Kannon Sheinman, and Maurice William.

[2] Oral Hygiene Committee of Greater New York: *Journal of Dental Research*, 1930, x, p. 497; October. (2) George T. Palmer: *Ibid.*, p. 655; December. (3) Oral Hygiene Committee: *Ibid.*, 1931, xi, p. 1; February. (4) George T. Palmer et al.: *Ibid.*, p. 7, February.

to find that the American Child Health Association, seeking to promote the public welfare and regarding dental decay as a 'serious problem,' has subjected dental caries to a special study. We note with great interest, also, that this important disorder is one of the first to receive attention in the Association's series of Monographs. Much that the Monograph presents is well known. We have no reason to doubt the validity of the mathematical processes described and applied, nor of the calculations presented, as relating to the particular records in the Monograph. But we are regretfully obliged to take exception to a number of the interpretations and deductions, because they conflict with reality, scientific knowledge, and clinical experience. . . . . " Our discussion is continued in the same spirit. Two members of the Sub-committee that prepared the original protest and this response were founders and are active members of the International Association for Dental Research, are editors of the *Journal of Dental Research,* and for years have been actively engaged in research in dental relationships. The need for penetrating research in all aspects of health service, including dentistry as one of its divisions, is everywhere understood. But data and conclusions published as fruits of research must bear close examination, and cannot be accepted if they fail to withstand analysis. We agree with one of Dr. Palmer's associates who has written: "Progress in science has never been achieved by accepting procedures without questioning the evidence." Until a research has been amply verified, its results are fairly open to question. Elsewhere in this issue (p. 299) appears this comment: "The dental profession has not been careful enough to reserve acceptance of novel statements and new methods until they have gone through the fire of the most thorough investigation and research."

Dr. Palmer's reply indicates, in conformity with assurances in Monograph III, that the Monograph was intended "to develop helpful information for the administrators of public dental clinics such as school clinics" (p. 8), most of whom are laymen. The Monograph was published in the name of a national organization of which the President of the United States is honorary president. The Association commands public confidence and respect, and its publications are evaluated accordingly. The obligation resting upon the representatives of such an association, to refrain from needlessly injuring any accredited

public agency, is self-evident. Numerous statements in the Monograph indicate that laymen were included in its anticipated sphere of influence. The Monograph was distributed widely among laymen. Dentists have noted its effect upon many lay readers.

II. MISREPRESENTATION IN MONOGRAPH III ABOUT "PROPAGANDA"

Outstanding among the conditions that made it impossible to regard Monograph III as a sincere presentation was its author's assertion, on p. 2, that "we are . . . . faced with propaganda directed at public health administration" claiming that *a policy of filling deciduous teeth protects the occlusal surfaces of first permanent molars against decay.* This invention of propaganda was accompanied by a pretentious array of data purporting to show that the propaganda was unjustified. By publicly knocking down the straw man thus set up, the author of Monograph III conferred upon himself an award of dependability, as an interpreter, that was impressive for those who did not see the straw. The implications from this theatrical "knock-out" tended to destroy the confidence of public-health administrators in public-health-education propaganda as actually conducted by dental organizations.

In Dr. Palmer's reply it was admitted (p. 14) that our objection to this performance was valid, and that the alleged propaganda never existed. But instead of expressing frankly an earnest desire to rectify a gross misrepresentation, Dr. Palmer's reply buried the disclaimer so deeply among misleading intimations relating to it that only the most careful readers recognize the fact that the pretended "propaganda" has actually been disavowed. This situation illustrates the characteristic in the Monograph to which the dental profession most emphatically objects; namely, the desire, willingness, or tendency to disparage dental practice rather than to present pertinent facts. The five citations in Dr. Palmer's reply, intended to show that on this matter probably the Monograph was not seriously in error (A-E, pp. 14–16), are not only evasive and irrelevant, but could not have been presented with any expectation that the admission of the author's invention of the "propaganda" would be clearly revealed. Of these citations, A, B, C, and D are plainly so general in scope as to encompass many conditions that affect the development and maintenance of sound permanent teeth. But they do not refer to the only

question involved: Does the filling of deciduous teeth protect the *occlusal surfaces* of first permanent molars against decay? Citation E, which is indefinite enough to seem to apply, refers to a paper by Dr. Marion R. Stevens,[3] and includes Dr. Palmer's assertion (p. 16) that "one of the author's conclusions favors prompt attention to the first permanent molars because of the relation of caries in the deciduous teeth to a parallel condition in the first permanent molars." One wonders why, instead of this vague statement, there was no quotation from Dr. Stevens' paper. But when, instead of relying upon Dr. Palmer's interpretation, Dr. Stevens' paper is examined, the reason for this omission becomes very evident. We find that the implication in Dr. Palmer's reply is not supported, as may be seen from the following quotation from Dr. Stevens' paper:

"Thus, from the foregoing methods, it appears that a very high correlation tends to exist between the degree of decay of the deciduous teeth and the structural formation of the first permanent molar. Having established this fact, *it is possible to deduce the conclusion that the degree of decay of the deciduous teeth has a mutual relation to the structural enamel formation of the first permanent molar; that good deciduous teeth and good first permanent molars, that poor deciduous teeth and poor first permanent molars, tend to be associated. This does not mean that one condition may be the cause of the other, but it does mean that some factor or factors tend to produce relatively similar conditions in deciduous teeth and first permanent molars, and that we cannot expect to have better first permanent molars until we have better deciduous teeth.*" [Italic in the original—page 396.]

From this quotation we see that Dr. Stevens has not said, as Dr. Palmer's reply would have the reader believe, that the filling of decayed deciduous teeth prevents decay in the *occlusal surfaces* of first permanent molars. There is nothing of the kind, and citation E is not only evasive but palpably misleading. Dr. Stevens referred to structural correlations that are not affected by fillings.

Among the practical applications of the conclusions of Dr. Stevens' study that bear on our discussion, but which Dr. Palmer's reply did not mention, was this significant allusion by Dr. Stevens to *prevention:*

[3] Stevens: "Significance of caries in deciduous teeth in relation to structural formation of enamel of first permanent molar at time of eruption." *Dental Cosmos*, 1927, lxix, p. 389; April.

"First permanent molars must be carefully examined for structural defects *as soon as* they erupt and whatever preventive measures are necessary [must] be employed *at once.*" [All was italic in the original.] (Page 396.)

On page 10 of Dr. Palmer's reply it is said that the Monograph "is openly clothed in the language of research, a language that is precise, that addresses itself to the points at issue." Was the invention of propaganda a "point at issue;" was it "precise?" In disavowing the propaganda, did Dr. Palmer's reply "address itself to the point at issue," and was it "precise?"

III. DESTRUCTIVE POSITION OF MONOGRAPH III

A. Typical statements in the Monograph

On pages viii and ix of the Foreword in the Monograph we read that "in the twenty-year period of development of school health work the public has been won over to its support. Our concern now should be for the justification of that support," etc. The Monograph "is contributory to . . . . determining the efficacy of one phase [dental service] of the procedures which make up the school health program." This statement of a constructive purpose suggests an attempt to realize it. One looks in vain in the Monograph, however, for any comment that directly encourages support for dental health-work in public schools. This was a destructive position for the Monograph to take, and has been widely regarded as such.

In our original protest we indicated a belief that Monograph III was destructive "in intent and presentation." The invention of the "propaganda" referred to above, and the elaborate means taken to show that this "propaganda" was unjustified, demonstrated that the author of the Monograph was unconcerned about the effects of false implications against the intelligence and integrity of the dental profession; also that he was indifferent to the prospective creation of the impression, upon lay minds, that there is no need to treat decay in deciduous teeth. If the author of the Monograph was ignorant of the dental realities in this connection, or careless about them, his presentation of this phase of the subject was none the less destructive.

It is difficult to find in the Monograph a statement indicating that any phase of dental practice is useful in public schools, but disparaging

statements protrude from every chapter. An author who goes far out of his way to invent propaganda, in order to show in detail its falsity, cannot reasonably plead that he has adhered so closely to the routine requirements of a technical description that he could not be expected to include qualifying statements intended to prevent misinterpretation of a matter-of-fact presentation. This applies especially to a publication intended for the guidance of laymen. The author of the Monograph introduced destructive comment where no justification could have prompted it. On pages 54 and 55 of the Monograph, to illustrate, laymen learn that "cleaning teeth as it is done by the dental hygienist . . . . cannot be expected to protect the child against caries." Laymen can hardly be expected to discover the gratuitous nature of this conclusion. A lay reader naturally infers from the statement that neither he himself nor any child can be more successful than a dental hygienist in cleaning the teeth, and therefore wonders why responsible and respectable dentists advise the use of dentifrices and tooth brushes for protection against decay. If the findings on which the conclusion in the Monograph about the futility of "cleaning teeth as it is done by the dental hygienist" were reliable, all would bow to the truth, for the facts must determine in every relationship. But the author did not study the procedure he disparages, and certainly had no reason to pretend to know anything about its adequacy. In a footnote in the Monograph, on p. 54, apparently as an afterthought, is this astonishing reservation: "We did not use iodine disclosing solution which limits the value of our measure of stain." Lay readers will not understand that here the word "limits" is a biometric euphemism for *vitiates*, nor will they comprehend that the many other deficiencies in the study of deposits on teeth, as shown in the Monograph and mentioned on pages 510–11 of our original protest, render the assertion in the Monograph, about the non-protective value of "cleaning teeth," entirely incompetent and irrelevant.

Several pages of the Monograph were filled with data demonstrating the obvious fact that the filling of a cavity in, say, an upper left first molar does not prevent decay in a lower right first molar. Lay readers easily acquire the idea, from a reading of this section, that dentists have been ignorantly filling cavities in teeth on the right side

to prevent decay in teeth on the left, and vice versa. The same lay readers conclude that the author of Monograph III has shown another deficiency in the dental profession.

On page 25 of the Monograph, at the *beginning* of Chapter III, it is said: "From a public health point of view the question is, 'What does dental work as now generally available contribute to public health?'" By "public health" we understand *public health.* On page 39, at the *end* of Chapter III, we read the author's answer in his conclusion that "present dental correction is palliative and not either preventive or curative. . . . . A real preventive program awaits solution of the problems of predisposition and cause." He does not suggest that *public health* is thereby promoted. Lay readers understand that "palliative" means mitigating or alleviating, as of a pain—nothing more—and conclude, from the use of this word, that the author of Monograph III has shown that there is no prevention of disease in present dental service. Laymen, understanding "real" to mean genuine and actual, are here told that genuine prevention is not being attained, although various physiological procedures, and a number of operations, in dentistry, are actually preventive of decay and infection, and their sequelae. If by "real" the author intended to suggest *perfect* or *ideal* prevention, not only of decay but also of imperfect enamel development and structure, and of all other inadequacies in teeth and their environment relating to decay that more knowledge might rectify, he did not by careful phraseology protect the realities of present dental practice against misunderstanding and disparagement. We regard the quoted conclusion above as a direct intimation that this "palliative" work, as in the case of "cleaning teeth," is not useful enough to be included in a school health program.

On page 8 of Dr. Palmer's reply it is said that Monograph III was intended "to develop helpful information for the administrators of public dental clinics such as school clinics." We understand that this is equivalent to saying it was intended to aid, to improve, to build up. The Oral Hygiene Committee, having accepted this assurance in the Monograph, criticized the latter as having failed to present constructive suggestions. We read with surprise, therefore, on page 20 of Dr. Palmer's reply, the following disavowal of the purpose expressed on page 8 and in the Monograph: "We are criticized for not trying to

show in our Monograph what should and could be done in a public dental health program. The Monograph made no attempt to deal with this question, not because it is unimportant but because this Monograph was not devoted to that subject."

Monograph III is entitled: "Public *health* aspects of dental decay in children." If we were asked to present published statements of the reasons why dental service should be included in a school *health* program, it would not be possible to select anything from Monograph III that would state this need clearly or persuasively to a lay board of directors. We know of no publication other than Monograph III that would so easily mislead a lay school board into deciding that, until "the problems of predisposition and cause" will have been completely solved as the basis for "a real preventive program," both the cleaning and the filling of teeth should be excluded from its *health* service.

B. Constructive statements in Dr. Palmer's reply

Having indicated in the foregoing subsection (A) several of the many reasons for regarding Monograph III as "destructive in intent and presentation," we assemble below some quotations from Dr. Palmer's reply bearing on this particular aspect of the discussions. We are glad to call special attention to these statements because they indicate constructive intent; by their contrast with the indifference shown in the Monograph, they emphasize the validity of our protest; and, having been made for the Association in response to public criticism, they presumably represent deliberation.

"The American Child Health Association realizes that misunderstandings have arisen due to . . . . shortcomings in appreciating the viewpoint of the dental profession." (Page 7.)

"We believe that oral disease, especially root-end infection, caries, and the premature loss of both deciduous and permanent teeth are serious handicaps to the health of children." (Pages 9–10.)

"We believed that the teeth of school children should be cared for as a health measure; that there was a real need for the various studies presented in Monograph III; and that these studies, even though necessarily limited in their scope, would play an important rôle in setting up a public dental program." (Page 10.)

"We are criticized for drawing the conclusion from our data that 'present dental correction is palliative and not either preventive or curative,' and that 'a real preventive program awaits solution of the problems of predisposition and cause.' . . . . This conclusion . . . . has reference to prevention in the strict sense, that is, the prevention of caries as a disease problem. It does not deny the value of 'extension for prevention' as a practice of operative dentistry, nor does it refer to the effect of recurrent decay. Obviously it supports the practice of early filling of caries to prevent its deeper penetration. By palliative was meant the lessening, abating or mitigating effect of fillings upon caries. Palliative was used to mean conservation." (Page 16.)

"The Committee . . . . refers . . . . to the argument for prophylactic odontotomy as follows (page 532): 'Studies of the teeth of adults have indicated the surfaces, and the specific locations on each surface, where dental decay commonly occurs early in life. These data suggest the nature, extent, and applicability of a practical preventive procedure.' The Committee cannot refrain from again asserting that 'Monograph III has not shown that these current views in dentistry are unfounded.' We agree. Monograph III had no intention of treating this subject." (Pages 42–43.)

"Does the Committee mean that much good has come from the dental health services established by schools, meaning reparative work, the encouragement of mouth cleanliness and instruction in diet? If this is what the Committee means then we agree. We too believe that this type of work has done much good. . . . . If the Committee means to imply that the American Child Health Association is not interested in a dental health program in the schools, the Committee is mistaken. This is a matter in which the American Child Health Association is intensely interested." (Page 44.)

These clarifying quotations from Dr. Palmer's reply lead us to believe that, when the Association sponsors its further publications on dental service, the realities of dental practice, both private and public, will receive the attention they merit.

IV. MATTERS RELATING TO THE UNRELIABILITY OF THE MONOGRAPH

If personal comfort were a paramount concern, we should discontinue this discussion with the last preceding paragraph. We continue to a logical conclusion in the interest of the broadest public considerations. We are persuaded that this is desirable by the following comment by one of Dr. Palmer's associates: "One lively debate over the

evidence will do more to clarify the truth and to provide an enthusiastic faith to carry forward that truth than years of claims, propaganda and persuasion without critical attacks."

A. Does the Monograph describe the technical procedures so definitely that other investigators can exactly duplicate them?

On pages 510–11 of our original protest, we discussed various aspects of the fact that the Monograph does not present a detailed statement of the procedure by which various dental data were obtained. Dr. Palmer's reply (pp. 24 and 25) stated that in this we were mistaken, but we were not shown where one may find a statement of the successive steps taken in the examinations, *so that they could be repeated exactly in a check upon the recorded observations and findings.* A photograph of the instruments in a surgical cabinet, bearing a legend about the general use of the instruments, is not a description of a particular surgical operation, nor would such a photograph and legend indicate exactly how an operator should perform it. In Dr. Palmer's "preliminary comment," last December, it was stated (page 656) that the Monograph was "published as a strictly technical presentation of the findings of a research." Strictly technical presentations of the findings of research contain either detailed descriptions of the methods employed, or references to these means as previously described, not only to indicate exactly how the work was done, but also to help other investigators to follow the procedures *in all their sequences and peculiarities*, or expertly to evaluate them.

Dr. Palmer's reply (p. 25) refers to a footnote in the Monograph which states that the examinations were made and the data "gathered by two dental hygienists." In the succeeding paper (p. 335), Dr. Dwyer calls attention to the fact that the dental hygienists did all this without competent dental supervision. Dr. Hyatt authorizes us to say that when he was consulted, he suggested that two dentists be associated with the study described in the Monograph, but was told that two medical men, two trained nurses, and two dental hygienists could do the work satisfactorily for those in charge of it. On page 25 of Dr. Palmer's reply, statements were made regarding Dr. Hyatt's advice on procedure that we are authorized to extend. When the subject of the different sizes of enamel defects was discussed, Dr. Hyatt

approved the basis of distinction for Type 1 only, and suggested the use of copper wires of different diameters to determine the sizes of the openings into defects.

B. Were a sufficient number of teeth examined for enamel defects to warrant the conclusions in Monograph III?

In our original protest, in discussing comment in the Monograph on enamel defects, we said: "These opinions . . . . allude . . . . not to the *same children at successive periods of observation* from age six to age thirteen, nor to *all* of the first molars in the children examined, but instead to different children examined once individually, and only to the so-called 'healthy' or 'non-carious' first molars remaining in a few of these children at the time of examination" (p. 515). "Table 4 indicates, in a very striking manner, that such a study as that recorded in Monograph III cannot give a true insight into dental conditions in particular age-groups *unless it deals with a very large number of children in each such group, or with the same groups of children examined recurrently through a succession of years*" (italic not in the original—p. 536). In a more direct objection to the fact that broad conclusions were based upon slight evidence, we wrote: "The Monograph fails to indicate why the author selected only about 12 per cent of the children and only about 25 per cent of the non-carious first molars to support his conclusions in Chapter IV regarding enamel defects and their relation to decay" (p. 519). We have therefore read with surprise, in Dr. Palmer's reply (p. 34), "that data on pits and fissures were not recorded [in the Monograph] for the 7500 children of the main study, but were limited to about 1000 children of ages 6 to 13 in the supplementary study." *Statements in the Monograph misled us* into believing, however, that more data had been available, but were not used. Many other readers have drawn the same conclusion for the same reasons. Nowhere in the chapter on enamel defects (IV) are there any recorded exceptions to the general statements on procedure in the Foreword, and in Chapter I. Accordingly, the reader seems to be told in the Monograph that the enamel defects had been noted in all of the non-carious first permanent molars in each of the 7500 children examined in 1927–28 and in all such teeth in all the "other groups of children"—number not indicated—from whom "supple-

mentary data were also obtained" (Foreword, p. vii). There is no statement in Chapter IV to the effect that the children indicated in that chapter on enamel defects were not selected from those designated in the Foreword and Chapter I.

On page 34 of Dr. Palmer's reply it was said: "When the Oral Hygiene Committee discovered this apparent discrepancy, two possible explanations might have occurred to them. One was that we did not possess the data on pits and fissures of the missing children; the other, that we were in possession of the figures but voluntarily concealed them. The Committee chose the latter conclusion." We chose the latter conclusion only because the language of the Monograph misled us into it. Having observed the unscientific tendency of the author of the Monograph in his *invention* of "propaganda" (page 307), an apparent selection of data seemed to accord with a similar intention. Shortly after the presentation of the Sub-committee's report at a meeting of the Oral Hygiene Committee, on June 26, 1930, we learned privately not what Dr. Palmer's reply indicates on page 34, but only that "the *study*" of pits and fissures was "confined to the children examined in New York." This we understood to mean that although the defects in the non-carious first molars in all of the other children were *recorded and known*, as indicated in the Monograph, only *the available data relating to New York Children were "studied;"* that is, prepared for the record *as published*. This private information seemed to emphasize the selection we believed had been indicated by the Monograph. Other collateral information supported this belief. In a press notice issued by Dr. Palmer, in March 1930, it was said that the Monograph "deals with analyses of mouth records of 7500 children of the 5th and 6th grades [ages 10–12] in many cities of the country as well as of several thousand children of lower ages." There is no intimation here that one of the most important aspects of the study described in the Monograph did not apply to all of the children indicated. Yet on page 34 of Dr. Palmer's reply, it is said that "the actual number of cases that were examined for pits and fissures [ages 6 to 13 inclusive] was 1199." In the tables in the Monograph (Tables 18–20) where the data have been assembled, and from which broad deductions have been drawn, the "number of children involved" is only 978, these having 2732 "non-carious" teeth (con-

taining per tooth an average of 0.7+ defect of Type 3 "considered as caries") for ages 6 to 13, an average of only 341 such teeth and 122 children for each age. The Monograph affects the externalities of a very precise and reliable mathematical presentation. But in a matter so elementary as that of clearly and directly noting exceptions to general statements of procedure, so that the reader would be *guided* to the truth, one is permitted to believe that general statements regarding "7500 children," and "several thousand children" in addition, apply where only 1199 were "examined" and only 978 were "involved." Although indifferent references in the Monograph to procedure misled us, the clarification of this situation has given special emphasis to the essential matter in the discussion on this point; which is that the number of children and teeth "involved" was not sufficient to warrant the sweeping conclusions based upon them in the Monograph. Our criticism in this relation was directed at the *selection* of a few data from a supposedly large number; now it applies automatically to the inadequacy of the small number of children and teeth *examined*.

In the summaries in the Appendix of our protest, we "used only children [Table 4, there] in which all first permanent molars are in place" because we sought to follow the plan indicated in the Monograph, allusions in the Foreword and Chapter I having misled us into the belief that this was the procedure followed in the work there described. On page 46 of Dr. Palmer's reply we are told that if we had used the data otherwise, the import would have been different—a further indication of the accommodating quality of the data. On page 39, Dr. Palmer's reply expressed the belief that all of the protested data in the Monograph are reliable. On page 46, agreeing with us in another relation, his reply stated that "data on 115 non-carious molars are certainly not sufficient to determine the relative liability of defective and defect-free molars to decay." Turning to the Monograph, however, one finds that only 173 such molars for age 13 (having an average per tooth of 0.64 defect of Type 3—"considered as caries") are included in the important Tables 18, 19, and 20, which are presented there as reliable. In the issue of the *Journal of Dental Research* containing Dr. Palmer's reply—in the discussion of a paper by Pollia on the unreliability of laboratory findings relating to focal infection, we note the following comment by Foster (p. 111): "Two hundred cases is

not enough to prove anything. . . . . A scientific therapeutist, a century ago, said it took ten thousand cases to prove a principle. Dr. Osler used to say that the worst thing about quackery was that it often cures."

C. Did chance affect any of the conclusions in the Monograph?

In Dr. Palmer's reply (pp. 35–36) there is an allusion to an "inaccurate graph," on page 520 of our protest, "that does not pass through the points that should be represented." The linear device we used to point out, in a simple way, a fall and a rise in percentage figures was obviously not a graph nor intended to be one. Discussing these percentage data [Table 18 in the Monograph], we said (p. 520): "If the author's data here are reliable for the deductions he offers, they also indicate that the percentage of first molars free from *detected* defect decreased [from 34 per cent] to a lowest value [16 per cent] at age 10, but thereafter rose steadily to a new maximum [25 per cent]. Whether this particular curve is significant, or merely an indication of error, variation, or coincidence, the author does not suggest." The doubts expressed in the last preceding sentence are removed by the following two contradictory assertions on pages 35 and 36 of Dr. Palmer's reply:

"The proportion of non-carious molars free from recognizable pits and fissures [as given in the Monograph] is less at the older ages than at the younger ages within the range of 6 to 13 years"—the only range considered (p. 35). [Here the figures are accredited by Dr. Palmer as reliable.]

"It may be shown by recognized statistical procedures that the difference that occurs between the percentage at 10 years of age and the percentage at 13 years of age [as given in the Monograph] is not greater than might occur by chance" (p. 36). [Here the same figures are discredited by Dr. Palmer as unreliable.]

If, as Dr. Palmer's reply indicated, "the difference that occurs between the percentage at 10 years of age [16 per cent] and the percentage at 13 years of age [25 per cent] is not greater than might occur by chance," it is obvious also that the significance of the entire series of percentages might have been nullified by chance, and that the conclusions in the Monograph based upon them are as unreliable as we suggested in our original protest; and as we continue to believe.

In another relation Dr. Palmer's reply emphasized the influence of chance on the figures in Table 18 of the Monograph (percentages of non-carious molars free from detected enamel defect). On pages 521–22 of our original protest "we give the calculated distribution of first molars at the same three ages [10, 11 and 12], per hundred of the maximum number," based in part upon the data in Table 18 of the Monograph. Tentatively we suggested that these results seemed "to show that the 'healthy' teeth *having observable enamel defects* were *not* 'more apt' than the others to *'escape caries'* [as the author of Monograph III stated], but instead more apt to *become carious*. If," we continued, "the author's data carry the import he ascribes to them, which we doubt, then he should not have ignored the fact that such examples as the foregoing indicate mathematically the direct opposite of his opinion as expressed in Quotation 9." In Dr. Palmer's reply our calculations for ages 10 to 12 were confirmed, and similar data for ages 6 to 9, and for 13, were added (p. 37). Dr. Palmer indicates, however, that the data "for ages prior to ten" would, on the same terms, *show the reverse* of the indication from the data for the ages after ten. It is said in Dr. Palmer's reply that in our tabulation for the years 10–12 we were "aided by a chance deviation" (p. 36). But this is a necessary admission that half of the data under discussion supports a conclusion in one direction, and that the other half supports a conclusion in the opposite direction. Both cannot be right. Who can tell which is wrong? On page 39 of Dr. Palmer's reply we read that "reliability" in the "biometric interpretation" means "that any random half of the records give the same conclusion as any other random half." How much has chance affected everything else in the Monograph? Here we are reminded that in a recent address, Chief Justice Hughes, speaking from experience, stated:[4]

"I regret to say that for the most part I find that the present tables of statistics of the work of the Federal courts are of very little use. We must be on our guard that in our passion for statistics we do not exhaust our efforts in obtaining extensive compilations of slight value. Those who deal with the furnishing of statistics are always in danger of being satisfied with a parade of figures."

[4] Editorial: "Hughes on statistics," *New York Sun*, February 27, 1931.

In editorial comment on these remarks it was said in part: "There are few men who know better how to employ statistics, and the statistical method in arriving at important conclusions, than does Justice Hughes. This is because he knows when to take his figures and when to let them alone—a gift rarer than many suppose. . . . . Statistics are all right, when used with discretion. But the public needs more warnings from men like Chief Justice Hughes against the danger of statistical indiscretion."

D. Miscellaneous matters relating to reliability

On page 515 of our original protest, commenting on Monograph records of enamel defects in non-carious first molars (Types 1–3), we indicated doubt as to the reliability of the data. But for purposes of analysis, having as we said "temporarily" assumed that the data were reliable, we then expressed the opinion that the values as recorded "should have *suggested*" the "*possibility*" of a progression from defects that were (*a*) not recognizable, to defects (*b*) succesively of Types 1, 2, and 3, and to (*c*) caries. We said that "nothing in Monograph III shows that this *possibility* was actually tested as a mathematical alternative." In this connection we noted "that the number of defects increases in sequence from Type 1 to Type 3 not only *horizontally* for a given age . . . . but also when followed diagonally *downward* to the right *in time* for *successive ages*," as shown in *Table 1*. At conferences of our Sub-committee, in June, 1930, we discussed the fact that, when the data were also followed diagonally *upward*, a similar numerical progression was suggested. But, as this would have ignored the factor of time, we derived no biological suggestion from it. Disregarding both our emphasis on the unreliability of the data in the Monograph and the fact that we suggested only a "possibility," Dr. Palmer's reply stated (p. 32) that the numerical indication of progression is the same whether the figures are followed diagonally downward or upward—which we had ourselves discussed; that we are "mistaken" in our "interpretation;" and that, using our "reasoning" but following the figures diagonally upward, one "may say that instead of pits and fissures becoming progressively larger the older the child is, they really become [*sic*] progressively larger the younger the child is." We do not believe that statistics may entirely replace judgment. In our biometric ignorance we do not see how something having attained age 13, let us say, can "*become*" anything at age 10. We understand that

"become" means to change or pass from one state of existence to another—to come to be something different. By using the same reasoning and language as that in Dr. Palmer's reply, "one may say" that by *reversing* a complete moving picture of a man emerging from a moving aeroplane two miles up, and jumping to his death, we should see how he looked when he ascended to the plane and reëntered it. In the picture, the ascension would show as clearly as the fall, but

TABLE 1

Data intended to show suggested "possibilities" of progression in the types of enamel defects mentioned in Monograph III

AGE	ENAMEL DEFECTS[1]		
	Type 1	Type 2	Type 3[2]
11	0.28	0.48	0.78
12	0.35	0.50	0.67
13	0.19	0.39	0.64

[1] Average total number of each type per "healthy" first molar (our protest, pp. 516–17).

[2] "May be considered as caries" (Monograph, pp. 44, 45, 76, 77).

would the ascension exhibit anything having any *biological* significance whatever?

In Dr. Palmer's reply it was suggested (p. 46) that the data in the Appendix of our original protest indicate "both frequent occurrence of teeth that have no observable enamel defects, and large liability of these teeth to caries." This is an allusion to the observation that 47.7 per cent of the non-carious first molars that had been free from *recognized* enamel defects in the first examination "became carious, or

were extracted . . . . *before* the *second* examination." But the following qualifications in our presentation of these results do not seem to have been noted in Dr. Palmer's reply.

(a) "Whether these teeth contained enamel defects that were overlooked at the first examination [for the Monograph] and became carious before the second [thirteen months later], cannot be determined, but, in 8 'O. K.' [non-carious and non-defective] molars that remained non-carious, 14 pits and fissures were noted at the second examination" (p. 537). [Were they overlooked at the first examination or did they become recognizable meanwhile?]

(b) "There is no reason to believe that the second examination was conducted less carefully or accurately than the first examnation" (p. 539). [Even if only half the number of obvious inaccuracies were due to error on the first examination, these errors render the whole series of data on this subject in the Monograph doubtful in validity.]

In our original protest we wrote: "The deep fog or Pickwickian atmosphere in which much of the work was done, and the Monograph written, is indicated in many places, but is well illustrated by the yes-and-no allusions to the significance of pits and fissures of 'Type 3,' [which]. . . . 'may be considered as caries'. . . . Just how *caries* can exist in a tooth while that tooth is *non-carious* we are unable to imagine, but possibly such problems can easily be solved with the aid of mathematical equations" (pp. 511–12). In commenting on this and associated contradictions, Dr. Palmer's reply presented this evasion (pp. 27–28): "Those openings. . . . with 'softening of the base or walls of the enamel' were classified arbitrarily as Type 3, *while possibly bordering on caries*. We therefore treated the data for each type [1, 2 and 3] separately so that conclusions may be formed if they are considered as enamel defects or if they are considered as caries. [Only defects of Type 3 were referred to in the Monograph as caries.] Regardless of the decision the conclusions are identical. This is the explanation [*sic*] of our expression, 'Type 3 (pit or fissure) *may* be considered as caries' (italic not in the original)." Here there is a labored effort to explain away contradictions. But in giving it, the authors evidently forgot all about another explanation on page 45 of the Monograph, as follows: "Type 3 pit and Type 3 fissure may be considered as caries. *This modifies conclusions to be drawn*

from average amount of enamel defect by age as presented in Table 19" [average number of enamel defects—Types 1, 2 and 3—in *non-carious* first molars at different ages; italic not in the original]. If Type 3 was not *actually* "considered as caries," how could it have "modified conclusions?"

Reliability of statistics is not automatic. It is an old saying that "although figures do not lie, liars may figure." We quote the following from *Tidings*, published by the Massachusetts Department of Public Health, Division of Child Hygiene (January–February, 1931) as fully expressive of our views on reliability:

"We must remember that the results arrived at by statistical methods are no sounder than the original data themselves. The original observations must mean what they purport to mean, and must be obtained under known conditions, if the conclusions drawn from them are to be valid." (Page 2.)

"We must emphasize the point that uncritical use of statistical methods involves a real danger because of the false impression of authority given by results mathematically expressed. Its misuse may even go so far as to bring the method into disrepute." (Page 3.)

V. REMAINING CHIEF ISSUE IN THE DISCUSSION (TREATMENT OF ENAMEL DEFECTS)

In Section II we indicated some of the reasons for our belief that the Monograph was not a sincere compilation. In Section III we stated some of the reasons for regarding the Monograph as "destructive in intent and presentation." In Section IV matters affecting the reliability of the Monograph were discussed. The remaining chief issue is the problem, in public dental clinics, of the treatment of enamel defects. On page 19 of Dr. Palmer's reply we are asked three questions that have been focussed upon this issue. Below we number these questions, italicize them for convenient differentiation, and answer them directly and separately.

1. *"Does the Oral Hygiene Committee favor a public clinic policy of cutting out and filling all the developmental grooves in the occlusal surfaces of first molars?"*

This policy has never been advocated, for private practice or for clinic service, by either the Committee or any of its members. There are two kinds of grooves. In the production of crown-forms before

the eruption of a tooth there are several separate parts known as primary lobes. When, having grown to their full size, these lobes meet and coalesce, the external indications of their union are called "developmental *lines*." The depressions on these developmental lines are named "developmental *grooves*." Other grooves, known as "supplemental grooves," are unimportant in this discussion because they are very rarely the sites of decay. Most developmental lines as a group, and as a rule all developmental lines at particular conformations on many surfaces—approximately 68 per cent of the total number—are practically free from recognizable defects and do not become carious.

In Dr. Palmer's reply we find this statement on page 21: "This idea [widespread practice of cutting out and filling all occlusal developmental grooves] was suggested to us by Dr. Thaddeus P. Hyatt." Dr. Hyatt authorizes us to state that he has "never by word, lecture, or written article advocated the filling of all *developmental* grooves, or of all *occlusal* developmental grooves."

2. "*Does the Oral Hygiene Committee favor only the cutting and filling of the recognizable non-carious enamel defects as soon as the tooth is erupted?*"

We favor (a) the "cutting and filling" of all "recognizable non-carious enamel defects" (b) as soon as possible after eruption of a tooth containing them, *but only when indicated by existing conditions*—(c) until research provides conclusive information that may make it possible to prevent the occurrence of enamel defects, or otherwise to prevent decay in such defects. See the answer to question 3, below, where this response is amplified.[5]

3. "*If only the latter policy is favored* [*Question 2*], *should this policy be applied only to private practice conditions and such other conditions as are suitable for experiment and research, or should the policy be extended to public clinics so as to be sure to reach great numbers of children who have non-carious enamel defects?*"

[5] This answer is restricted to the question, but the Oral Hygiene Committee also "favors" the procedure now in force under the auspices of the New York Department of Health (Annual Report, 1928, p. 49): "(1) Prophylactic cleaning and recording defects, (2) removal of unsavable teeth, (3) operative care for pre-carious pits and fissures, (4) filling of carious cavities."

In both private practice and in public clinics, a suitable procedure for the prevention of decay should be applied to all recognizable non-carious enamel defects as soon as possible after eruption of the teeth containing them.[6] This preventive procedure in a public clinic should be *operative* or *advisory*.[7] By this we mean that if for any *unavoidable* reason, such as inadequate financial resources, a public clinic cannot apply a suitable *operative* preventive procedure[8] to each recognizable enamel defect, the clinic, so far as its resources permit, should, as an *advisory* alternative, (a) locate and record for each patient all the recognizable enamel defects; and (b) give to the patient, a parent, or a guardian the facts of this examination, (c) so that the patient may be encouraged and helped to consult a dentist as soon as possible regarding prompt protection against decay in the enamel defects disclosed.[9]

Referring to the work that "can be carried out practically in a public clinic" Dr. Palmer's reply contains this statement (p. 20): "The large number served requires rapid and routine methods and little variation of procedure to meet individual needs." Although we do not favor any policy in a clinic that would disregard in the

[6] In this connection, some dentists use the word "*eradicate*" to mean enlargement with a finishing bur and polishing to insure physiological cleansing during mastication, thus removing the food-trap that defects usually present. Some dentists also use the word "*immunize*" to mean protection against decay, in a defect, with the aid of chemical treatment. The latter, because it is not lasting in its effect, is not now being employed to any great extent. We refer to these two words to clarify our comment, not to endorse these uses of them.

[7] In 1928 this policy was instituted in the Dental Division of the New York Department of Health. We understand that it is still in effect.

[8] An "operative preventive procedure" applied to an enamel defect, as we use the phrase, may be an instrumental process of (a) polishing or enlarging, or both, to insure free movement of saliva and food along the bottom and sides of a defect that does not extend to the dentin or too close to it; or (b) of "cutting out" the defect, and filling the ensuing cavity.

[9] As to the importance of advisory service in the work of a public clinic, we quote the following pertinent comment from Dr. Palmer's reply (p. 18): "Even in periodic health examination clinics the advice in regard to 'lumps, swellings, and tumors' is to refer the case to a surgeon" [outside of the clinic]. *The New Zealand School Dental Service* (1928) contains the following comment (p. 13): " . . . . Children are examined when they enter school, and parents are advised of any defect present. Where for any reason they do not desire to obtain treatment from a private practitioner, the work will be undertaken at the school dental clinic."

slightest degree the special interest of any individual, it happens that the treatment of enamel defects as proposed above lends itself exceptionally well to a routine that would include relatively rapid procedure for a comparatively large number of patients. The simplicity, directness, uniformity, and economy of the suggested procedure emphasize this opinion. The advisory alternative, in direct helpfulness and educational value, is included in this judgment. A policy of caring for the teeth of pre-school and kindergarten children, on operative preventive lines—*while we endeavor to gain information that would make all operative procedures unnecessary*—would result not only in saving money, time, and trouble for the child, but also in conserving its teeth and health. We do not suggest that an impossible program should be attempted. It stands to reason, however, that the work of every public clinic should be brought to the highest possible degree of efficiency consistent with professional responsibility, understanding, and capacity, and with *obtainable* public financial support. It is a part of the duty of responsible health servants to show cause why this attainment is required in the public interest. On this aspect of the subject we commented in our original protest in part as follows (p. 506): "The Monograph does not aim to show what should and could be done, in a public dental-health program, for the protection of teeth against decay, nor does it present constructive criticisms of current deficiencies in funds or personnel, or otherwise, which, in the public interest, should be overcome, and which such criticism might help to rectify. Instead, the author seems to believe that such an effort would not be practical. Apparently in his opinion, *recommendations* for a public-school health policy should suggest not plans for the most complete and effective protection of child health, but merely anything less that would be conveniently available under prevailing conditions, whatever they might happen to be. In this view, a policy passable today would also be acceptable years hence—nothing should be done to effect improvement; things as they are might be worse; adopt the easiest way, not the most desirable method." The description of a research that included in the Monograph the *invention* of "propaganda" and an elaborate exposure of the falsity in that propaganda—all with destructive intent—might reasonably have indicated also a constructive policy on closely related aspects of the stated subject of the Monograph.

In Dr. Palmer's reply we read (p. 33): "In any public dental health examination made at a public clinic, there would very likely be many pits and fissures so small that they *would not be detected* and these are just as likely to become carious as those that *are detected*. Since they are not discovered they would not receive the alleged benefits of prophylactic odontotomy were such a program in force." In our original protest we said, on this subject (p. 508): "Some enamel defects may be so minute that they cannot be easily detected, but their proportion is so small, compared with the larger number that can be, that *it would be inexcusable to do nothing about those that can be found.* Not every enamel defect becomes carious, but the majority that will certainly soon harbor decay is so large that, to prevent caries in these, an operative preventive procedure relating to them is *necessary where health is the primary consideration*" (italic not in the original). On the same page in Dr. Palmer's reply (p. 33), the following related comment appears: "It is important therefore to find some means of identifying all pits and fissures that are likely to become carious before it may be considered as proper policy to advise prophylactic odontotomy in public clinics. And this is the main point which we believe the Oral Hygiene Committee failed to recognize." To which we reply that from the humanitarian point of view, to say nothing of common sense, it would be inexcusable to do nothing about the many defects that are *likely* to become carious until we can perfectly and ideally identify those that certainly *will not* become carious. The desirability of devising a perfect method for an ideal differentiation between the enamel defects that certainly will become carious and those that surely will not is so obvious and well recognized that we wonder how Dr. Palmer and his associates could "believe" that a committee consisting of practising dentists would not be unanimously aware of it. In Dr. Palmer's reply it is said (p. 8) that the Monograph "is a contribution to diagnostic method, not to etiology." We notice with regret that, despite the emphasis in Dr. Palmer's reply on the need for a method of perfect differentiation, Monograph III has contributed nothing to the development of a differential diagnostic procedure.

On page 8 of Dr. Palmer's reply similar emphasis on the obvious is made to appear, in these words, like an issue: "We contend [*sic*] that a method of distinguishing pre-carious enamel defects is much needed.

We believe that there is a great deal of support of this thesis amongst the dental profession. The Oral Hygiene Committee does not meet this contention. . . . ." We know of no "contention" among *dentists* about this obvious need, and wonder why laymen should "contend" about it. If dentists had been among the staff that conducted the research described in the Monograph, they would neither have tainted its biometric perfection nor reduced its public value in any respect, but they certainly would have helped to make it more useful for dentists. Possibly the time is near at hand when certain physicians, doctors of public health, and other health workers will be less complacent about their superior understandings, and less confident in their condescensions regarding a profession against which they show traditional prejudice. Let us hope that, in a better spirit, they may be more ready to accept the fact that dental education in American universities prepares dentists for effective and honorable participation in those phases of health service for which as dentists they have been particularly and responsibly trained. Dentists understand that dentistry is quite as imperfect and just as much in need of research as is any other division of health service. Dentists are contributing earnestly and effectively to the progress of dental research, to the betterment of dental practice, and to the improvement of health service in general.

The "remaining chief issue in the discussion" may be summed up in a few words. Monograph III and Dr. Palmer's reply indicate a belief that, in a public clinic, nothing should be done to protect the individual patient against the decay that inevitably arises in many enamel defects until research makes it possible to distinguish between the defects that certainly will decay and the defects that certainly will not decay. Our Committee adheres to the view that, *until research makes it possible completely to prevent the occurrence of enamel defects, or provides a perfect differential diagnostic procedure,* all recognizable enamel defects should, in a public clinic, be suitably treated as soon as possible because most of these defects will certainly decay. Our Committee would do what is possible now to protect the health of the patient against the consequence of the decay that otherwise will certainly arise. Monograph III and Dr. Palmer and his associates, noting that some defects do not decay and being unable to distinguish

them, would do nothing now about any of them, but instead would wait for the development of a differential method of diagnosis that may never be devised. One policy, by being positive, contributes directly to the conservation of teeth and the protection of health; the other policy, by being negative, endangers teeth and health. Which of these two opposing policies is to the best interest of the public?

VI. SUPPLEMENTARY COMMENT

A comparison of Dr. Palmer's reply with our original protest reveals many additional disagreements, only a few of the most important of which are referred to briefly below. Readers who may be interested in details should compare all disagreements in their original forms. In this expectation, quotations above and below have been reduced to a minimum. The page numeral at the beginning of each paragraph indicates the location of the subject in Dr. Palmer's reply.

Pages 7 and 10. Our references to lack of sincerity related to the Monograph, written by one person named on the title page as its author. We assumed that the statements in the Monograph were made by its accredited author, not for him. We did not assume that the members of the American Child Health Association as a body passed on the merits of the Monograph.

Pages 10 and 13. Genuine research, which is welcome everywhere in dentistry, cannot be destructive of truth. But what is often called research may be as destructive and as worthless as inability, or insincerity, or indifference can make it. We do not regard Monograph III as a gift from heaven, or necessarily true because it was published. We do not believe that the American Child Health Association wishes any one to assume that the Monograph must be right because it cannot be wrong. Like all publications of research, that described in the Monograph cannot be accepted as conclusive before its findings are verified. Meanwhile, data hitherto accepted as true, with which the Monograph disagrees, may remain true.

Page 17. Nowhere in our protests have we used the term "prophylactic odontotomy." On page 325 we indicate that by "operative preventive procedure" we have referred to more than one variety of intervention. The very frequent intrusions of "prophylactic odonto-

tomy" into Dr. Palmer's reply indicate prejudice against this procedure, although it is claimed that the author of the Monograph did not "evaluate" it.[10]

Page 22. Dr. Palmer's reply assures us that "medicine has been misled too many times by clinical experience for us to rely upon it as final proof." It may be said, quite as appropriately, that medicine has also been misled too many times by the results of laboratory experiments and biometric devices for us to rely upon either as final proof.

Page 22. We are asked to present evidence of the ability of members of the Oral Hygiene Committee to "recognize dangerous enamel defects." This can be done in a practical demonstration, at recurrent examinations of the same children. All observable enamel defects on certain surfaces are dangerous, in the sense that they are apt to become carious. The Committee has not suggested that anything can be "recognized" which cannot be "observed."

Page 24. The principle in our reference to vaccination has been ignored. Vaccination of all persons in a community to prevent the death of those persons for whom small-pox would be fatal—without any knowledge of the immunity or predisposition of the individual person—is an acceptable protective procedure. On the same principle, an operative preventive treatment applied to all recognizable enamel defects in a mouth, to prevent the decay of those defects that would soon become carious (loss of teeth, pulp infection, and their sequelae) —without any knowledge of the immunity or predisposition of the individual tooth (enamel defect)—is an acceptable protective procedure. Treatment of all in a community, of persons or of teeth (defects), to reduce mortality in the unknown number that can thus be protected, is the common principle in these two means of prevention. We repeat what we said in our original protest in another connection (p. 527): "Mortality is a decisive factor. When the mortality is high, it is desirable to apply a preventive procedure to all, regardless of the probable immunity of some. The same principle applies to prevention of loss of first molars, especially because of their outstanding

[10] See comment by Dr. Hyatt on this subject: *Journal of Dental Research*, 1931, xi, p. 335 (succeeding paper, this issue).—[*Ed.*]

importance in mouth *health*, and in the normal development of the jaws and head."

Pages 26–27. It is said that in our original protest we pointed out that, in the Monograph, the "method of detecting stain was faulty" and that inasmuch as the "records on stain, tartar and gingivitis were admittedly not as dependable" as the "measures of caries," the Monograph "should not have reported upon them at all." This does not correctly state our position. We said (pp. 510–11) that because the *work* on stain was inadequate, this fact should have been recognized and the whole chapter on this subject omitted as the conclusions were "incompetent and irrelevant." Dr. Palmer's reply expresses inability to understand why, "if a result does not turn out as reliable as we should like it to be, then reference to such material should be omitted." Those who in their statistical enthusiasms are, in the words of Chief Justice Hughes, "satisfied with a parade of figures" (p. 319), may not readily see why worthless data in a record are like drunken men in a procession. Dr. Palmer pretends to find an inconsistency in our position on this point, and our objection to the selection of and emphasis upon *preferred* data to the exclusion of data that "prove something contrary to predetermined conclusions" (p. 519). To us the two devices seem to be "two of a kind," both untruthful and reprehensible.

Page 28. We learn that "protective" defects are protective in a biometrickian sense, not in a common sense. Last summer one of our number noted the consequences of two leaks in the roof of a cottage. During a heavy rain, water poured through one old leak; it dropped occasionally through a new one. The larger leak did the most damage but it proved to be more "protective" than the smaller one, for it induced the owner to have the roof repaired without further delay.

Page 30. Monograph III seeks to correct and replace the published results of past researches. Therefore its methods should have been notably better and more reliable than those with which former results were obtained. The results to which we have alluded as challenging the validity of data in the Monograph were obtained in the same general manner as those given in the Monograph; but, by representing

a very large number of persons and teeth, the earlier data are plainly more reliable than those in the Monograph.

Page 38. The two contrasted quotations from our original protest may mislead the reader, who should compare the related comment in the original. One of the two quotations reads as follows: "There is no evidence in the Monograph of any recurrent examination of the same children and teeth to justify these conclusions, which in the main *appear to indicate predictions* rather than observed conditions" (p. 507). In this quotation, "these conclusions" referred directly to unjustified inferences in the Monograph. "These conclusions" also presented predictions which, in the positive and extreme forms stated, could be warranted only by the results of recurrent examinations of particular teeth. Such examinations were not described in the Monograph. We have objected to the assumptions in Monograph III that all of the observable enamel defects in the "non-carious" first molars at, say, age 13 were also present as observable defects in the same first molars of the same children at age 6, and that none of these observable defects became carious during the seven years from age 6 to age 13. We suggested that a possible progression in defects—from unobservable microscopic defects through "Types 1, 2 and 3" to caries—caused the average numbers of observed defects from age 6 to age 13 to become as uniform as they appear in Table 19 in the Monograph. The author of Monograph III did not eliminate the possibility of progression in enamel defects. This possibility cannot be disposed of by any dialectic procedure. Until it is tested by examinations of the same teeth in the same children at successive ages, the data in Monograph III relating to average number of observable enamel defects per tooth, from age 6 to age 13, will obviously bear a doubtful significance.

Page 39. Here there is an effort to persuade the reader to conclude that there are no contradictions in three quotations from the Monograph, which we called "contradictory assumptions." It is said in Dr. Palmer's reply that, of the following two of the three quotations contrasted on page 524 of our original protest, the first "is simply a conservative reiteration" of the second:

(a) "A tooth *without such defects* as we are able to observe is *just as likely to become carious* as are the others (italic not in the original)." (Monograph, p. 43.)

(b) "First molars which seemed to our observers to have *no enamel defects* were *more liable to caries* than were those which could be described by them in terms of pit and fissure (italic not in the original)." (Monograph, p. 48.)

By directly comparing these two sentences, after their reduction to common terms, we note the following:

(a) Teeth . . . no obs. defects . . . *just as* likely . . . carious . . . have obs. defects
(b) Teeth . . . no obs. defects . . . *more* likely . . . . carious . . . have obs. defects

According to Dr. Palmer, "just as likely" is simply a "conservative" way of saying "more likely." Such comment about these two obviously discordant references is equivalent to saying that the expression "x *equals* y" is simply a "conservative" way of stating that "x is *more* than y."

Pages 41–42. We fully "appreciated the meaning of a correlation." Dr. Palmer's reply seems to disregard our statement that "although the surfaces of all the teeth are subject in common to destructive influences, *local conditions* decisively aid, or make it possible for, these *influences* to *cause* decay" (p. 530). As to the influence of "accidental combinations of circumstances," we note that there was no discussion of the effects of some common ones that we suggested as being influential.

Page 43. We appreciate the complimentary references to our playfulness and humor, but the pleasantry in Dr. Palmer's reply does not distract our attention from the issue, which, in our protest, we indicated in this way (p. 533): "On page 39 the author [of the Monograph] states the obvious as follows: 'The chances for further development of caries in any one tooth are smaller when previous decay in that tooth has been corrected.' But if decay is not dependent upon *local* factors [as is contended in the Monograph], and is 'a function [peculiarity] of the individual organism and not the tooth,' how can this be true? Possibly the author [of the Monograph] would wish us to rely upon this brilliant comment on page 29 of the Monograph: 'Some common factor exists in mouths *at certain times* which exposes all of the teeth present to likelihood of decay. *Such waves of influence could conceivably be checked by filling the cavities*' (italic not in the original)." Then we asked, in effect, how, as the author of the Mono-

graph claims, can these waves of *general influence* be "checked" by repair of the *local damage* they are doing? There is no answer in Dr. Palmer's reply. We must accept the conclusion as a biometric revelation—as an overpowering biological deduction from numerical data in the Monograph. It proves beyond all possible doubt the automatic sureness of the mathematical processes in the Monograph. It is deserving of world acclaim. Think of it! The "waves of influence" along a shore that is being devastated during a storm can be "checked" by quickly repairing the damage they are doing, *while* they are doing it! How wonderful is biometricks!

VII. SUMMARY

Section II: An invention of propaganda in Monograph III is discussed, and its disavowal noted and emphasized.

Section III: The destructive character of Monograph III is illustrated. Assembled quotations from Dr. Palmer's reply, indicating a constructive position that was not shown in the Monograph, foreshadow a fair presentation of dental subjects in future publications by the American Child Health Association.

Section IV: Evidences of the unreliability of the data in the Monograph, and also the misleading character of some of its presentations, are discussed.

Section V: The treatment, in public clinics, of enamel defects—the remaining chief issue in the discussion—is considered, and the position of the Oral Hygiene Committee restated.

Section VI: A few additional disagreements with statements in Dr. Palmer's reply, of many that might be noted, are briefly indicated.

CORRECTIONS OF STATEMENTS BY DR. PALMER AND ASSOCIATES RELATING TO RESEARCH MONOGRAPH III OF THE AMERICAN CHILD HEALTH ASSOCIATION[1]

THADDEUS P. HYATT, D.D.S., F.A.C.D., AND H. SHIRLEY DWYER, D.D.S.
New York City

I. LETTER FROM DR. THADDEUS P. HYATT

While I greatly regret the circumstances that make it necessary for me to respond to a personal accusation, I feel it is incumbent on me to correct remarks in Dr. Palmer's reply[2] concerning my disapproval of Monograph III. Dr. Palmer stated (p. 26) that I 'questioned' results presented in the Monograph "*because* they did not support *his* [*my*] *own views* regarding prophylactic odontotomy" (italic not in the original). This statement is not true. My objections to Monograph III were, and still are, *chiefly* those indicated briefly below:

(1) I object to the implication that my profession ever engaged in or encouraged any propaganda, as mentioned on page 2, which claimed that a policy of filling deciduous teeth protects the occlusal surfaces of first permanent molars against decay.

(2) I regard it as absurd to devote pages to a mathematical demonstration of the obvious fact that the filling of one tooth does not prevent decay in another tooth on the other side of the mouth. This information (?), *following* the statement made on page 2 regarding active propaganda, would lead any person unacquainted with dentistry to infer that a dentist fills a tooth on the left side in the belief that it prevents a tooth on the right side from decaying. While Monograph III does not state this in so many words, the lay reader is encouraged by the comment to think so. There is nothing in the Monograph to indicate the contrary.

(3) I object to the implication in the Foreword (last paragraph) which misleads the reader into believing that the dental profession or many of its leading members approved the Monograph in its entirety. This is not true, because the dentists to whom the proof of Monograph

[1] Separate letters to the *Journal of Dental Research*, by Drs. Hyatt and Dwyer, have been combined in one article.—[*Ed.*].

[2] Palmer et al.: *Journal of Dental Research*, 1931, xi, p. 7.

III was submitted were practically unanimous in advising against its publication until further investigations could be made. Although this advice by many leading dentists was rejected, there is no indication of this fact in the Monograph. Instead it is intimated in the Foreword that dental opinion was generally favorable.

(4) While destructively criticizing every phase of the dental activities investigated, nothing constructive was suggested as to what should be done or what might be done. *There is not one word in Monograph III advocating any phase of dental service*, despite the fact that the reader naturally expects, from its title, to find evidence on the effect of dental service upon the *health* and welfare of the child.

I have been informed, from most trustworthy sources that, before the publication of Monograph III, it was stated that a portion of the study was intended to refute the idea of "prophylactic odontotomy." This purpose was not stated in the Monograph, where all could have seen it. No research is truly scientific if it is *intended* to "get" anything except the truth. Any such destructive *purpose* would disregard facts that did not help to attain the predetermined end.

Dr. Palmer's criticism of my motive in objecting to Monograph III is false, and would make me appear to be bigoted, narrow-minded, and prejudiced. In the forty years in which I have served my profession, and the public through it, no one has ever accused me of these vices. I believe that Dr. Palmer and his associates did not fully realize the implications of their unjust accusation.

II. LETTER FROM DR. H. SHIRLEY DWYER

I was surprised to find, in Dr. Palmer's reply,[2] some personal references to a purely impersonal situation. I am quoted as being perfectly satisfied with the accuracy of the observations of his hygienists.

Lest this should lead to false impressions as to the reliability of Monograph III, I wish to say that this statement was made by me *while the hygienists were in training*, not after they began their examinations in the field, where they were *not supervised by any person having competent dental knowledge*. I trust that the quotations from the comment of others, in Dr. Palmer's reply, are not as deleted, abbreviated, or misapplied, as those relating to me.

Quotations Relating to "Dentistry or Stomatology"

THIS JOURNAL uses the term "dentistry" to signify the science and art of oral health service in all its local and systemic relationships. The meanings of words, which are not necessarily restricted to *etymological* significance, acquire expanded or new import in accordance with convenience and usage. Thus the term "medicine," which originally meant to remedy or cure, now also means *to make remedial or curative treatment unnecessary*. "Stomatology" is a synonym for "dentistry." We prefer the older term because its use respects both the history of dentistry and the efforts of those who have brought dentistry to its present status.—*Jour. of Dental Research*, 1929, ix, p. iii.

If dentistry were merely dental mechanics, a two-year course in dental technology, based on graduation from a high school and conducted along lines similar to those for the education of optometrists, would probably afford sufficient training for dental practice, and oral health-service could be reserved for a new specialty to include oral surgery, which might be named stomatology and made analogous to ophthalmology and other specialties of medicine. But with dentistry developed into the *full service equivalence* of an oral specialty of medicine, the creation of a medical specialty of stomatology would be redundant—and there would be no occasion to substitute "stomatology" for the name established by custom.—*Carnegie Foundation's Bulletin on Dental Education*, 1926, p. 135.

The assumption that dentistry would be something more or better than it is, if all or a part of it were called "stomatology," exemplifies a readiness to attach more importance to labels than to contents, or more significance to make-believe than to reality. The term "medicine" originally meant to remedy or cure. ["*Medicina* (ars)"—healing art; "*medicina* (res)"—a remedy; "*medicus*"—of healing; also a physician.] Today "medicine," by including measures to *prevent* sickness and by throwing *etymology* overboard, means not only to remedy or cure, but also *quite the contrary*—to make remedial or curative treatment unnecessary. As medical knowledge has advanced and medical procedures have been changed in consonance, including the development of prevention and of surgery, new meanings have been imparted to the term "medicine." But instead of replacing "medicine" by a succession of words intended to indicate *etymologically* the broader or deeper modern meanings, the import of the original term has been conveniently enlarged as the scope of medical practice has expanded. Thus, the term "medicine" has come to mean more and more as *physicians*, without restricting themselves to its *etymology*, have included *more and more in its significance*. Along the same line of reasoning one sees clearly that, although *etymologically* the term "dentistry" refers only to the teeth, nevertheless logically it also comprehends the contiguous parts and their systemic relationships. Certainly by *usage* in conformity with this logic, and also with the criteria of common sense, the term "dentistry" has *acquired* the broader meaning suggested by the more detailed and lengthy term "oral health-service." By logic, usage, and convenience, the term "dentistry" has thus become the *practical* equivalent of "stomatology," which *etymologically* means the sum of scientific knowledge concerning the mouth, but *etymologically does not include the rest of the body* (although "stomatology" is now *used* by some to include systemic relationships with the mouth). This being true, we are unable to agree that "dentistry" and "dentists," which are terms that have long represented the earnest and aspiring endeavors of generations of faithful practitioners of oral health-service, should be formally replaced for *etymological* reasons by "stomatology" and "stomatologists."—*American College of Dentists*, March, 1929.

The New York Academy of Dentistry . . . without any diminution of its abiding purpose to promote intimate association and cooperation between dentists and the practitioners of all other kinds of health-service, believes that, in the interest of the patient, dentistry should be *continued* as a separately organized profession, based on systems of ethics, education, jurisprudence, and journalism that will fully meet all public needs at each stage of its evolution.—*N. Y. Academy of Dentistry, April, 1929.*

The Dental Society of the State of New York . . . expresses the earnest conviction . . . that the most effectual development of oral health-service will be insured by the *continuance* of dentistry as a separately organized profession . . . [and] also records its unabated desire and intention to promote personal and professional accord, cooperation, and mutual helpfulness between dentists and the practitioners of medicine and all other kinds of health-service.—*Dental Society of the State of New York, May, 1929.*

INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH

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